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LXXIX

THE MECHANISM OF DEGLUTITION (SECOND STAGE)
AS REVEALED BY CINE-RADIOGRAPHY

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The mechanism of swallowing remains a physiologic problem difficult of analysis largely because of the speed with which the bolus descends from the level of the fauces to the esophagus. William Harvey, comparing the "quickness" of the motions of the heart chambers to that of deglutition, wrote in his immortal *De Motu Cordis*:

"All these events because of their quickness seem to occur simultaneously in the twinkling of an eye. Likewise in swallowing: lifting the tongue and pressing the mouth forces the food to the throat, the larynx and the epiglottis are closed by their own muscles, the gullet rises and opens its mouth like a sac, and receiving the bolus forces it down by its transverse and longitudinal muscles. All these diverse movements, carried out by different organs, are done so smoothly and regularly that they seem to be a single movement and action, which we call swallowing."¹

From the Divisions of Anatomy, Surgery, Radiology and Otorhinolaryngology of the University of California Medical School.

The substance of this paper was presented at the annual meeting of The American Laryngological, Rhinological and Otolological Society, May 25, 1950, San Francisco, California.

To Magendie (1813) we owe the classical division of the mechanism of deglutition into stages: (a) a voluntary stage during which the contracting of the mylohyoid and the longitudinal lingual muscles enables the tongue to propel the bolus past the fauces; (b) an involuntary "almost convulsive" contraction of the pharyngeal constrictors during laryngeal elevation which forces the material into the upper end of the esophagus; (c) a slower, involuntary stage during which the bolus is driven along into the stomach by the esophageal circular fibers.²

However in 1880 Kronecker and Falk³ suggested that fluids and semi-fluids were projected directly into the stomach by the contraction of the mouth muscles (mainly the mylohyoid muscles) before pharyngeal and esophageal muscular contraction occurred; and that these latter contractions took place after the bulk of the bolus had passed in order to sweep along any remaining particles. Kronecker reported that with the contraction of the mylohyoid muscles at the beginning of deglutition there was a rapid rise to a manometric pressure of 20 cm of water in the posterior mouth, pharynx, and upper esophagus, a pressure presumably sufficient to force semi-fluids through the esophagus to the stomach without the aid of peristalsis. He and Meltzer introduced balloons into the pharynx and esophagus and recorded the passage of the peristaltic wave some time after the descent of ingested liquids.⁴ They later modified their views in that they discovered that fluids were not squirted directly into the stomach, but were detained just above the cardia where the after-coming peristaltic wave eventually swept them into the stomach.

Cannon (1898-1900) showed that the act of deglutition varied in different vertebrates and with different consistencies of materials ingested. He introduced the use of x-ray studies of the opaque (bismuth subnitrate) bolus. His observations were in agreement with Kronecker and Meltzer's theory that fluids in the human were shot to the level of the cardia mainly by the action of the mylohyoid muscles, and not by peristalsis. But he demonstrated that the swallowing of solids and semi-solids was quite different, being a relatively slower peristaltic-like process comparable to Magendie's earlier description. Cannon found that deglutition in fowls (whether fluids or solids) was comparatively slow and peristaltic, that there was no mylohyoid squirting action, and that in the absence of such action a greater reliance on gravity was present. He pointed out that when the mylohyoid muscles were denervated in the dog, fluids were no longer rapidly squirted into the esophagus; thereafter it was necessary for the animal to raise its head and swallow after the manner of a bird. In cats, though the movements of deglutition were some-

what more rapid than those of fowls, the process appeared to be one of peristalsis only. This passage of the bolus synchronously with adjacent pharyngeal and esophageal muscular contractions (i.e. peristaltic) was observed in the horse and man when ingesting solids and semi-solids; only fluids were squirted ahead of their peristalsis by the action of the mylohyoid muscles.⁵

Cannon believed that the solid bolus descended in the human, in general, as follows: when the food is masticated it is gathered in a depression on the dorsum of the tongue; the tip and lateral aspects of the tongue press against the hard palate and teeth to prevent escape of the bolus anteriorly or laterally into the mouth or cheeks. Respiration is reflexly suspended. Contraction of the mylohyoid and hyoglossus muscles (surely Cannon must have meant styloglossus) then forces the tongue superiorly and posteriorly driving the bolus against the soft palate and posterior pharyngeal wall. During this phase the action of the palatopharyngeus muscles draws the pharynx into a narrow cleft against which the levatores palati pull the soft palate and prevent entrance of the bolus into the nose. The esophagus now opens when the larynx rises. The epiglottis is pressed back until it shuts the laryngeal aperture⁶ and the bolus is driven into the esophagus.

By 1910 most of the physiological texts described deglutition in terms of mylohyoid and pharyngeal constrictor action, in which the epiglottis acted as a lid for the laryngeal opening.

However this epiglottic action had been disputed as early as 1892 when Anderson Stuart had reported a case of lateral pharyngotomy. He wrote that he observed that the epiglottis remained vertical during deglutition, appearing to be squeezed between the base of the tongue and the upper boundary of the larynx.⁷ Negus in 1927 cited Stuart's work as proof that the epiglottis had no function in swallowing; he concluded that "the epiglottis is an accessory olfactory organ;" that it preserves "the integrity of the olfactory sense when the mouth is open . . . by coaptation to a long soft palate, thereby preventing entrance of air by the mouth."⁸ Yet Mosher during the same year published his fluoroscopic studies of tongue, epiglottis and hyoid bone movements, observing that the epiglottis "does act as a cover for the larynx during swallowing."⁹

Several more or less marked points of difference from the bulk of previous physiological study were presented by the English radiologist Barclay in 1930.¹⁰ He denied the role of the pharyngeal constrictors: "there is no suggestion that the posterior pharyngeal wall comes forward at all or any other sign that indicates a constrict-

tor action of its muscles." He supported the theory that the epiglottis did not act as a laryngeal trapdoor; he described it as remaining vertical during swallowing, "like a rock under a waterfall." He believed that the principle factor in deglutition was the production of a negative pressure in the pharynx and upper esophagus, and that the bolus was thus sucked into the esophagus. Barclay had observed fluoroscopically that the pharyngeal airspace had appeared to be momentarily obliterated just before the food shot down the dorsum of the tongue. He postulated that this pharyngeal space was obliterated by (a) raising the larynx, (b) and retracting the tongue. This pharyngeal space was then opened up (while the outlets to nose, mouth and laryngeal aperture were closed) by (a) dropping the larynx; (b) and allowing the tongue to move forward, thus presumably creating a negative pressure. Anrep introduced a nasal catheter into the pharynx and calculated that a negative pressure of approximately 35 cm of water occurred apparently just before the descent of the bolus. Barclay did not entirely discount the role of muscular propulsive action; "although I hold that negative pressure is the main force, yet propulsive action is latent, ready to come into play if the negative pressure is not sufficient to accomplish the swallowing of the bolus."¹¹

Many of Barclay's conclusions gained rather wide acceptance in the following years. Negus in his Presidential Address to the Section of Laryngology of the Royal Society of Medicine in 1942 supported the suction theory; he reiterated that the epiglottis had no function in deglutition. However, he did point out that a contraction wave passed down the pharyngeal constrictors during swallowing.¹²

Most of the physiology texts today incorporate Barclay's and Negus' views. Best and Taylor (4th Ed.) quote Anrep's figure of a 35 cm water negative pressure as existing in the pharynx and upper esophagus (during Magendie's second stage), and "thus aiding in the descent of the bolus." Semi-solid food, however, is forced into the esophagus by the action of the pharyngeal constrictors. The (a) elevation of the larynx—not its closure by the epiglottis, which remains upright—and (b) the approximation of the vocal cords by the muscles attached to the arytenoid cartilages are thought to be the factors in preventing food from entering the larynx.¹³

Yet subsequent work has not always agreed with these conclusions. In 1939 Welin observed fluoroscopically that the epiglottis bent down during swallowing until all the contrast medium had passed; it then assumed its normal position "by way of a rapid turning back motion."¹⁴ He also described some cases of dysphagia due

to the slow return of the epiglottis to the erect position. In 1942 Johnstone also (fluoroscopically) noted that the epiglottis turned over, acting both as a chute for food and a guard for the larynx. He further observed that the posterior pharyngeal wall became quite thickened (on radiographs) by the contraction of the pharyngeal constrictors. Johnstone considered Barclay's theory of negative pressure to have no foundation; he pointed out that no suction had been noted in high esophagostomies.¹⁵

In a recent article Negus refuted Barclay's suction theory by citing a patient with a pharyngostome; during swallowing, a jet of water squirted out of this opening under a pressure of between 100 and 200 mm of water.¹⁶

NOMENCLATURE

Note should be made of the confusion in current literature regarding the term "pharynx." Frederic Wood Jones points out that the term should be used only in the sense of the older anatomists, i. e. the upper expanded portion of the digestive tube, between the esophagus below and the mouth and nasal cavities above. The soft palate in no way forms an incomplete septum between the mouth and pharynx; the soft palate in all mammals separates the pharynx from the nasal chambers. In no case should the term "pharynx" be extended to the posterior part of the nasal chambers; such terms as naso-pharynx, naso-pharyngeal isthmus are meaningless.¹⁷

METHODS

High speed cine-radiography is an essential prerequisite for the analysis of the second stage of deglutition, so rapid is the sequence of motion. Ordinary radiographic techniques such as fluoroscopy and the use of "spot" films are quite inadequate and have obviously proved misleading. Unless the observer is familiar with the various stages of deglutition as revealed by cine-radiography, the appearance seen in "spot" films may be incorrectly interpreted since their relationship to the cycle remains unknown.

In our earlier studies we employed a 16 mm motion picture camera equipped with an f.95 lens which photographed the fluoroscopic screen indirectly through a front surfaced mirror at the rate of 60 frames per second. The camera was synchronized with the fluoroscopic tube (60 cycles per second) by means of a $\frac{1}{4}$ h.p. 3 phase motor. The x-ray unit was operated at 140 KV, 30 ma. The films, which were of good quality, were studied by projection at various speeds. In addition, each frame was examined separately

under a magnification of 12 diameters to reveal detail and measure the serial changes.

In order to secure better visualization of detail, especially in the soft tissues, a further series of subjects was examined by cine-radiography employing the larger 35 mm film at the slower speed of 30 frames per second. The quality of the negatives obtained in the second series was far superior to that of the first. The apparatus consisted of a 35 mm Bell and Howell motion picture camera provided with a Biotar f.85 lens which was mounted to photograph the fluoroscopic screen (Patterson B2) directly at 30 frames per second. Linograph film, which is very sensitive to green light and capable of being devolved to reasonably high contrast, was found to be most satisfactory. The camera was synchronized with the fluoroscopic unit by means of a $\frac{1}{8}$ h.p. motor with a commutator which interrupted the transformer primary circuit so that x-rays were produced only during one-half of each revolution of the commutator. The Machlett tube (Dynamaxx 25 AA) was operated at 100 KV, 140 ma.

The subjects employed were normal adults, both male and female. In addition a few patients suffering from derangements of deglutition were examined in order to determine, if possible, the role of the constrictor muscles. The material swallowed was either thick or fluid barium, water or air (and presumably saliva). Since only with radio-opaque materials can the precise position of the bolus be determined, the protocols of the barium swallow only are given in detail. However, the sequence of motions in the cycle can as easily be determined when radio-lucent materials are employed. Cine-radiography at speeds slower than 30 frames per second is unsuitable for studies of deglutition.

Such factors in deglutition as (1) the descent of the bolus; (2) the movements of the epiglottis; (3) the sphincteric action of the laryngeal aditus; (4) the superior excursion of the larynx; (5) the action of the pharyngeal constrictors; and (6) the movements of the base of the tongue were thus readily observed. The time relationships involved were easily calculated inasmuch as each picture represented either $1/60$ or $1/30$ of a second.

REPORT OF CASES

Cine-radiographic studies at the rate of 60 frames per second.

CASE 1.—Male, 30 years of age. A thick barium bolus was given the subject.

(1) The leading edge of the bolus was observed to plunge from the level of the fauces to the level of the cricoid in $10/60$

second. Four frames or $4/60$ second after the bolus was noted at the fauces, the leading edge was seen on the posterior surface of the tongue. Thereafter at $1/60$ second intervals the bolus: (a) touched the epiglottis; (b) filled the valleculae; (c) entered the pyriform sinuses; (d) passed the level of the laryngeal aperture; (e) arrived at the level of the cricoid; (f) passed the cricoid level and entered the esophagus. One-third second later the superior end (or after-end) of the bolus had reached the esophagus, i.e. had passed the cricoid level. Approximately $20/60$ second later this superior end of the bolus was one inch below the level of the cricoid.

(2) Movements of the epiglottis during descent of the bolus were striking. The epiglottis remained in its upright "resting" position until the beginning backward thrust of the base of the tongue; at this same time ($24/60$ second before the bolus appeared at the fauces) the epiglottis began to arch posteriorly and inferiorly. Thus $3/60$ second after the bolus appeared at the fauces, the tip of the epiglottis touched the posterior pharyngeal wall making a 45° angle. One-twentieth of a second later the epiglottis appeared practically horizontal; $2/60$ second later it made a 45° angle below the horizontal; $1/60$ second later it was 60° below the horizontal; $1/60$ second later it was slightly in excess of 60° minus. During the ensuing $20/60$ second the epiglottis remained swung down at this angle; in fact, the subject appeared to have partially "swallowed his epiglottis." One-fifteenth second after the superior end of the bolus had reached the esophagus the epiglottis began its upward swing to its normal pre-deglutition resting position; $10/60$ second later it was 45° below the horizontal; at subsequent $1/60$ second intervals it was: (a) 15° below the horizontal; (b) horizontal; (c) in final upright resting position. Thus the epiglottis darted back from 45° below the horizontal to upright in $4/60$ second.

(3) The laryngeal aditus and vestibule airspace were obliterated shortly before and during the descent of the bolus. The details of this laryngeal sphincteric action were better seen in the 35 mm films and will be discussed in that section.

(4) Laryngeal excursion (or the upward movement of the thyroid cartilage and hyoid bone) began $54/60$ second before the leading edge of the bolus was noted at the fauces. Calculation of the extent of this movement was done by comparing the distances between the cricoid and an arbitrary point selected on the mandible. One-half second after the larynx began to rise it appeared to have reached its fullest superior excursion (although during the pharyngeal descent of the bolus it seemed a fraction higher). The larynx main-

tained this position while the superior end of the bolus reached the esophagus. One-third second later the larynx finally began to descend. It did not reach its predeglutition resting position until fully one second later.

(5) Observations of the posterior pharyngeal wall agreed, in the main, with Johnstone's findings of a rapid wave of pharyngeal constriction.¹⁸ One-sixtieth of a second after the leading edge of the bolus had passed the level of the cricoid (i.e. while the pharynx was filled with the after-coming bolus) a wave-like appearance was first seen on the posterior pharyngeal wall which then traveled inferiorly with the descent of the opaque mass. The progress of this wave could be followed for 30/60 second; it disappeared 6/60 second after the end of the bolus had passed the esophageal opening.

(6) The backward thrust of the base of the tongue was first noted 2/60 second after the larynx had reached its fullest superior excursion (or 24/60 second before the bolus appeared at the fauces). Also the soft palate was seen to press against the posterior pharyngeal wall at the time the bolus appeared at the fauces, and to remain there until the end of the bolus slipped into the esophageal opening.

CASE 2.—Male, 43 years of age. The patient had unilateral (left) IX, X, XI, XII cranial nerve paralyses following a bullet wound three years before. The bullet had entered just lateral to the corner of the mouth, presumably passed beneath the base of the skull in the region of the jugular foramen, then lodged beneath the skin lateral to the external occipital protuberance. He exhibited hemiatrophy of the tongue; unilateral vocal cord paralysis; sternomastoid and trapezius atrophy; and pharyngeal, palatine and lingual sensory changes. He had been unable to swallow since the time of injury, having taken atropine continuously to control salivation, and having gavigated himself several times daily for three years.

The patient was given 5 cc barium gruel and instructed to attempt to swallow it; he later was made to perform the Valsalva effort (maximum voluntary contraction of the expiratory muscles with the glottis closed) in order to demonstrate the flaccidity of the paralyzed left pharyngeal wall.

The pharynx appeared quite dilated; the flaccidity of the left pharyngeal wall largely accounted for this. Despite the repeated attempts of the patient to swallow (as seen by a number of superior excursions of the thyroid cartilage and hyoid bone), the pharynx remained dilated, and the mass of the barium came to lie just above the level of the cricoid. As the patient continued his efforts an oc-

casional segment of this barium would then appear to be tossed back up into the valleculae, only to trickle down the pyriform sinuses, and pool, as before, just above the cricoid. An occasional trickle of barium was seen to sweep over the aryepiglottic folds into the trachea; in no instance was barium seen to pass into the esophageal opening. The epiglottis was observed to bend to the horizontal position as with the weight of the bolus in the valleculae, but in no picture did the epiglottis touch the posterior pharyngeal wall, nor descend beyond the horizontal.

The time relationships during this partial descent of the bolus were markedly slower than in the normal. Thus it required fully 30/60 second for the leading edge of the bolus to pass from the filled valleculae to just above the cricoid (whereas in the normal the leading edge plunged from the level of the fauces to the level of the cricoid in 10/60 second). And even at this time the main mass of the barium appeared to be detained within the markedly dilated valleculae. One-fourth second later (or 15 times slower than in the normal) the bulk of the bolus appeared to have reached the pyriform sinuses. At 55/60 second later most of the barium had collected just above the cricoid level; at no time was barium seen to pass below this level. One and one-half seconds later (as the larynx descended) the patient was seen to project this barium pool upward into the pyriform sinuses; the remainder of his efforts consisted of the repeated shifting of the bolus between the pyriform sinuses and the cricopharyngeus level (at 1 sec. intervals) until the accumulating particles of barium passing over his aryepiglottic folds into his laryngeal aperture resulted in his "choking," and expelling the bolus through his nose and mouth.

In a separate photographic series the patient performed the Valsalva effort. The pharynx dilated to approximately three times normal diameter (i.e. compared to the normal subject performing the Valsalva). As the positive pressure increased within the pharynx the superior end of the esophagus was seen to open and a thin trickle of barium (residual from the above swallowing attempts) was observed to pass from the level of the cricopharyngeus into the esophagus, though the patient had been making no swallowing efforts.

Cine-radiographic studies at the rate of 30 frames per second.

A 35 mm Bell and Howell motion picture camera with a Biotar f.85 lens photographed the fluoroscopic screen (Patterson B2) at the rate of 30 frames per second using Linograph film. The camera was synchronized with the fluoroscopic unit by means of a 1/8 h.p.

motor with a commutator which interrupted the transformer primary circuit such that x-rays were produced only during one-half of each revolution of the commutator. The Machlett tube (Dynamaxx 25AA) was operated at 100 KV, 140 ma.

CASE 1.—Female (P. G.), 22 years of age. A thick barium bolus was given the subject.

(1) The leading edge of the bolus descended from the level of the inferior border of the mandible (that is, from about the level of the fauces) to the esophageal opening in 4/30 second. At 1/30 second intervals the bolus (a) filled the valleculae, (b) the pyriform sinuses, (c) and entered the esophagus. At 9/30 second after the first appearance of the bolus some of the barium retained in the valleculae began to separate from the main mass of the bolus, constituting a second smaller bolus which the subject swallowed during a second complete cycle. The after-end of the bolus entered the esophagus 7/30 second behind the leading edge. During the descent of the barium an airspace was noted to precede the bolus at all times.

(2) The epiglottis, coincident with the posterior-superior excursion of the base of the tongue, described a posterior-superior arc. Thus when the bolus appeared at the fauces the epiglottis was horizontal; with the descent of the barium the epiglottis moved well below the horizontal, remaining there until 4/30 second after the superior end of the bolus had passed into the esophagus. Thereafter the epiglottis resumed its upright position within 2/30 second.

(3) The sphincteric action about the laryngeal aditus was well visualized in these 35 mm films. Early in the swallowing cycle a shortening of the aryepiglottic folds occurred, manifested by a superior excursion of the arytenoid cartilages. Thus 12/30 second before the appearance of the bolus the arytenoids began rising, and the airspace outlining the laryngeal aditus and vestibule began diminishing. By 4/30 second later the arytenoids had moved superiorly the distance of one cervical vertebra and the aditus and vestibule were almost obliterated. At 1/30 second after the appearance of the bolus the vestibule airspace was found to be obliterated. Within 6/30 second following the descent of the after-end of the barium into the esophagus the aditus began to open. Some 4/30 seconds later the aditus and vestibule were about 50% their pre-deglutition volume, and the arytenoids had almost completely descended.

(4) The beginning of the upward excursion of the hyoid bone was noted 19/30 second before the leading edge of the bolus appeared

beneath the mandible. From its resting position opposite the distal quarter of the fourth cervical vertebra the hyoid rose to a point opposite the disc between the third and fourth cervical vertebrae by 12/30 second before the appearance of the bolus. During the descent of the bolus the hyoid rose just slightly higher (to the level of the distal quarter of C_3). Then 29/30 second after the leading edge of the bolus had entered the esophagus the hyoid began to descend, reaching its resting position 4/30 second later.

(5) A constrictor wave was first noted on the posterior pharyngeal wall 2/30 second after the leading edge of the barium had entered the esophagus. This peristaltic wave swept down the wall and disappeared within 12/30 second, or 7/30 second after the last barium had entered the esophagus.

(6) The posterior and superior thrust of the tongue (as shown by diminution of the airspace at the base of the tongue) was first observed 15/30 second before the appearance of the bolus. The tongue appeared to begin its anterior excursion (i.e. toward the pre-deglutition position) 6/30 second after the descent of the last barium.

CASE 2.—Female (W. L.), 27 years of age. A thick barium bolus was given the patient.

(1) The bolus descended from the fauces to the esophageal opening in 5/30 second. The after-end of the barium entered the esophagus 6/30 second later. An airspace was observed just ahead of the bolus at all times.

(2) The epiglottis was just above the horizontal when the bolus appeared at the fauces. Within 2/30 second the epiglottis bent down to 30° below the horizontal, remaining there until 1/30 second after the bolus had passed, when it resumed its upright position in 4/30 second.

(3) The laryngeal aditus and vestibule airspace began to diminish 8/30 second before the appearance of the bolus. By 2/30 second later this airspace was 50% diminished. At 1/30 second after the bolus had appeared the airspace was obliterated, remaining so until a little air appeared in the aditus 1/30 second after the last barium had entered the esophagus. Within 7/30 second thereafter the laryngeal airway had resumed its pre-deglutition volume.

(4) The hyoid bone was observed to begin rising 12/30 second before the appearance of the bolus. The maximum hyoid excursion was the height of one cervical vertebra (i.e., from a point opposite the distal quarter of C_4 to that of C_3); and it occurred during the descent of the bolus.

(5) No constriction wave was found on the posterior pharyngeal wall, presumably because the subject had just previously swallowed a portion of the barium (before the camera had been placed in synchronism), and the present bolus was thus too small to show details of the posterior pharyngeal wall.

(6) The beginning backward excursion of the tongue was noted 8/30 second before the appearance of the bolus. The onset of this posterior movement of the base of the tongue was coincident with the beginning of the posterior arc of the epiglottis.

CASE 3.—Male (J.), 30 years of age. A thick barium bolus was given the subject.

(1) The bolus dropped from the fauces to the esophageal opening in 10/30 second and the after-end followed 12/30 second later. A considerable amount of air was noted just ahead of the bolus in all of the frames.

(2) The epiglottis began to descend from its upright resting position 9/30 second before the bolus appeared at the fauces. It did not reach the horizontal until 7/30 second after the first barium was seen at the level of the mandible; then it swung to 60° below the horizontal within 4/30 second. The return of the epiglottis was not observed as the subject had swallowed relatively late during the film run and the automatic timer (set at 3 seconds) had shut off the fluoroscopic unit before the post-deglutition resting state had been reached.

(3) The laryngeal aditus began to diminish and the arytenoids to rise 8/30 second before the appearance of the bolus. The aditus and vestibule did not appear to be completely closed (i.e., no airspace) until 9/30 second after the bolus appeared at the fauces, or 1/30 second before the leading edge of the barium entered the esophagus. The laryngeal airspace began to reappear 3/30 second after the passage of the last barium into the esophagus.

(4) The hyoid began its ascent 27/30 second before the bolus appeared; it had reached its point of maximum excursion (not quite the height of one cervical vertebra) 1/30 second after the barium was first seen. The hyoid began descending 5/30 second after the last barium entered the esophagus.

(5) A peristaltic wave was first noted on the posterior pharyngeal wall 1/30 second after the leading edge of the barium had entered the esophagus; it traveled down the wall in 11/30 second, entering the esophagus synchronously with the after-end of the bolus.

(6) The backward thrust of the tongue was first observed 23/30 second before the barium appeared at the fauces.

CASE 4.—Male (R. J.), 26 years of age. A thick barium bolus was given the subject.

(1) The bolus descended from the fauces to the esophagus in 8/30 second, and the after-end followed 10/30 second later. Air was observed ahead of the barium in all frames.

(2) The epiglottis was 45° above the horizontal when barium appeared at the fauces. At 5/30 second later the epiglottis had reached its maximum arc of 30° below the horizontal, remaining in that position until the bolus passed. It then assumed its upright position within 2/30 second.

(3) The excursion of the arytenoids, and the sphincteric action of the laryngeal aditus and vestibule were complete 6/30 second after the bolus appeared. Air was seen in the aditus 6/30 second after the last barium passed into the esophagus; by 4/30 second later the aditus and vestibule were fully open.

(4) The hyoid began ascending 32/30 second before the appearance of the bolus, reaching its maximum excursion (the height of one cervical vertebra) 1/30 second after the barium was first noted.

(5) The posterior pharyngeal wall peristaltic wave was first seen 2/30 second after the leading edge of the barium had entered the esophagus; it moved down the wall in 8/30 second, entering the esophagus synchronously with the after-end of the barium.

(6) The backward excursion of the tongue was first noted 29/30 second before the appearance of barium at the fauces.

Studies also were done using very thick barium (i.e. solid bolus), very thin barium (fluid bolus), and water. Subjects also were studied who were instructed to swallow without any substance having been placed in the mouth, the bolus presumably consisting of air and saliva. The findings in these subjects were similar to those previously examined, except that with the solid bolus the pharyngeal constrictor wave appeared to accompany the leading edge of the bolus into the esophagus, whereas with the fluid bolus the wave was first noted after the leading edge of the barium had entered the esophagus.

The application of these normal findings to the interpretation of functional derangements of the swallowing act is illustrated by the following case:

CASE 5.—Female (M. M.), 50 years of age. In May, 1945 the patient had sustained a skull fracture and had thereafter developed signs of a right carotid cavernous sinus fistula. When she attempted to swallow solids she experienced choking sensations and complained that the bolus stuck in her pharynx; she frequently expelled it through her nose and mouth. Although the patient was able to swallow liquids and soft foods sufficiently well to maintain her nutrition, she continued to have sensations of retained food particles in her throat and frequent episodes of choking. On August, 1945 ligation of the right carotid artery and the accompanying vein was done. Signs of her fistula disappeared, but there was no improvement in her swallowing.

A thick barium bolus was given the patient.

When the leading edge of the bolus appeared at the fauces the pharyngeal airspace was found to be somewhat smaller than normal due to the partial posterior movement of the tongue. The hyoid bone was at its resting position opposite the upper quarter of the fourth cervical vertebra. The epiglottis was 60° above the horizontal. The laryngeal aditus was about $\frac{3}{4}$ open.

Within $1/30$ second the leading edge of the barium appeared just below the angle of the mandible. The further posterior excursion of the tongue almost obliterated the upper portion of the pharyngeal airspace. The epiglottis was about 30° above the horizontal. The hyoid had moved upward to the level of the intervertebral disc between the third and fourth cervical vertebrae. The aditus was $\frac{1}{2}$ closed.

By $1/30$ second later the pharyngeal airspace had completely disappeared, the posterior portion of the tongue being almost in contact with the dorsal pharyngeal wall. The epiglottis remained at 30° above the horizontal, and the hyoid at the level of the intervertebral disc between the third and fourth cervical vertebrae. The aditus was not quite $\frac{3}{4}$ closed. These relationships remained unchanged until $44/30$ second later (a markedly prolonged interval), when the tongue moved forward slightly, as manifested by the re-appearance of a small pharyngeal airspace.

At $9/30$ second later the leading edge of the bolus reached a point about halfway between the angle of the mandible and the level of the epiglottic tip; by $8/30$ second later it entered the valleculae. The epiglottis was found to be 30° above the horizontal at that point. By $2/30$ second later the aditus was $\frac{3}{4}$ closed, and the epiglottis slightly less than 30° above the horizontal. At $2/30$ sec-

ond later the barium flowed around the epiglottic tip; $1/30$ second later it reached the level of the aditus (which had completely closed $1/30$ second before); $1/30$ second later it was at the level of the cricoid; and $1/30$ second later it entered the esophagus.

Within $6/30$ second thereafter the main mass of the bolus began to divide into two segments. The superior segment occupied the valleculae and pyriform sinuses. At that point the epiglottis was noted to have dropped 45° below the horizontal. At $2/30$ second later the after-edge of the inferior segment of the barium entered the esophagus. By $2/30$ second later the base of the tongue began to move forward. Within $2/30$ second the epiglottis began its return from below the horizontal; at $1/30$ second later it was horizontal, and $1/30$ second later it had returned to its resting upright position. At that point the aditus was seen to be completely open. The superior segment of the bolus remained within the pharynx, the patient apparently beginning another swallowing cycle just as the film ended.

In no frame was any evidence seen of a peristaltic wave on the posterior pharyngeal wall.

The sequence and time intervals in additional studies with this patient swallowing (a) water and (b) air and saliva were almost identical with those found when using the barium bolus.

It was concluded that although these studies revealed a normal sequence of events, there was a considerable delay during the passage of the bolus through the pharynx. This delay, associated with the absence of a peristaltic wave on the posterior pharyngeal wall, suggested a partial paralysis, presumably unilateral, of the pharyngeal constrictor muscles.

DISCUSSION

The second stage of deglutition occurs with such extraordinary rapidity as to make it impossible, except in the grossest fashion, for the eye to follow and the mind to analyse the many almost simultaneous movements which constitute this functional mechanism. Thus, despite the numerous studies of the action of swallowing over the past century, opinion as to the mechanism is far from satisfactory and often as not flatly contradictory. Even such elementary matters as the role of the epiglottis remained unsettled. Although "spot" roentgenograms taken during the cycle are most useful, their interpretation is difficult and, indeed, has proved to be most misleading when attempts have been made to arrange films made from different cycles sequentially in order to demonstrate the mechanism. On the

other hand, the development of relatively high speed cine-radiography offers the most favorable opportunity of analysing details of the mechanism and the precise phase and sequence of the motions. However, we have found from experience that so rapid are the movements, minimum speeds of from $1/30$ to $1/60$ of a second are required to stop motion adequately. With slower speeds, such motions as the descent of the epiglottis and its return may be entirely missed since these movements frequently occur over less than $1/15$ second.

In the second stage of normal deglutition, the first objective sign of its onset is the ascent of the hyoid bone accompanied by that of the larynx. The lingual muscles have already rolled the bolus into position immediately anterior to the fauces in preparation for the propulsive effort of the tongue. The base of the tongue is then thrust rapidly backwards and upwards by the action of its intrinsic musculature, the mylohyoids, palatoglossus, and styloglossus muscles. Apart from the evidence obtained by cine-radiography, we have been able to observe directly the preliminary motions of the tongue by means of high-speed motion picture photography in a case in which the entire cheek on the left side had been resected as far posteriorly as the pillars of the fauces for malignant disease. Preparatory to swallowing, the intrinsic muscles of the base of the tongue contract to form a flat platform or piston-like plunger the dorsal surface of which is directed postero-superiorly. On deglutition, the plunger and with it the entire tongue suddenly ($1/20$ second) force the bolus into the fauces. At this stage the contraction of the styloglossus muscle is a prominent feature. It is this motion of the tongue which obliterates the upper portion of the air-shadow seen outlining the pharynx in lateral roentgenograms.

The backward and upward motion of the tongue is associated with and would seem to be directly responsible for the initial movement of the epiglottis. This structure is tilted somewhat dorsally from its almost vertical position so that the valleculae begin to open widely. It is assumed that this motion is transmitted by the contraction of the stylohyoid and hyoglossus muscles to the epiglottis through the hyoepiglottic ligament. The evidence suggests that the digastric muscles are responsible for the preliminary elevation of the hyoid bone; the arrangement of the intermediate tendon permitting the upper and backward movement of the hyoid, larynx and tongue on later contraction of the stylohyoid and styloglossus muscles.

As the bolus is projected beyond the fauces, the soft palate is pulled against the posterior pharyngeal wall by the action of the levator and tensor palati muscles. The closure of the aperture to the

nasal cavities is completed by the contraction of the palato-pharyngeus muscle establishing Passavant's ridge on the posterior pharyngeal wall. The pharyngeal constrictors would then seem to go into action giving rise to the peristaltic wave visualized in the films on the posterior wall. While it is apparent that the initial thrust of the tongue may be sufficient to project fluids as far as the esophageal opening before the constrictors come into play, it is certain that semifluid and solid boluses are propelled through the pharynx largely by the aid of these muscles. The cine-radiographic studies of cases with paralysis of the pharyngeal wall, both unilateral and bilateral, leave little doubt on the importance of the constrictors for deglutition. Hence, we are quite unable to agree with Barclay's contention that the descent of the bolus is mainly the result of the development of a negative intrapharyngeal pressure. This theory must be abandoned on the further score that the laryngeal aperture is still open and the pharynx in communication with the air of the lungs while the bolus is descending well into the pharynx. Such conditions render the development of a negative pressure impossible.

Regurgitation of the bolus into the nasal cavities during the period of constrictor contraction is prohibited not only by the approximation of the soft palate to the posterior pharyngeal wall but also by the continued backward and upward thrust of the tongue. Furthermore, the passage of the faucial pillars towards the mid-line serves to narrow the passage to the buccal cavity.

The action of the epiglottis as a trapdoor sealing the entrance to the larynx during swallowing is established unequivocally by cine-radiography. High-speeds of photography are necessary to demonstrate this movement. Frenckner¹⁰ was unable to obtain absolute evidence of this motion by cine-radiography for the obvious reason that his apparatus was inadequate. Laryngeal closure is further effected by the vigorous sphincteric action at the aditus and vestibule associated with further elevation of the larynx. As has been mentioned, the descent of the epiglottis begins simultaneously with the backward movement of the tongue. However, this movement is purely preparatory to the opening up of the valleculae which is mediated probably through glossoepiglottic attachments. The actual closure of the epiglottis does not occur until the bolus has reached the valleculae and is about to flow around its lateral borders into the pyriform sinuses. By this time the epiglottis is approximately horizontal supporting the bolus on its ventral surface. The final tipping of the epiglottis downwards occurs with extraordinary rapidity and delivers the bolus almost immediately to the crico-esophageal junction. This action proceeds a fraction prior to the appearance of the

pharyngeal contraction wave and is associated with the slight further postero-superior excursion of the larynx. It is, therefore, reasonable to suppose that the movement is initiated by the contraction of the stylopharyngeal muscles.

The descent of the epiglottis is intimately related to the sphincteric closure of the larynx and would seem to be controlled by both active and passive factors. Since the aryepiglottic folds may be observed to shorten, it is probable that the contained aryepiglottic muscles, as well as the thyro-epiglottic, assist in pulling the epiglottis backward and downward. At the same time the further elevation of the larynx projects the arytenoid eminence toward the base of the epiglottis which would serve to tilt that structure passively. Closure of the vestibule and approximation of the chords are doubtless due to the contraction of the thyro-arytenoid, vocal and related muscles. So vigorous is the constrictor mechanism that it alone is sufficient to prevent the ingress of ingested food into the larynx as has been reported in cases of excision of the epiglottis.

The return of the epiglottis to the upright position is so rapid as to suggest that it is affected by the elastic recoil of that structure and by the action of the hyoepiglottic ligament. However, it has been observed that its return is associated with the anterior excursion of the tongue which doubtless assists the movement.

SUMMARY

(1) A cine-radiographic technique is described by which human deglutition was photographed at the rate of 60 and 30 frames per second.

(2) Normal human deglutition involves essentially an initial rise of the larynx, a vigorous backward thrust of the base of the tongue, followed by a wave of pharyngeal constrictor muscle contraction.

(3) The piston-like action of the tongue may be sufficient to project fluids into the esophageal opening before contraction of the pharyngeal constrictors. Solids, however, are propelled through the pharynx mainly by these constrictors.

(4) The bolus is prevented from entering the larynx by the sphincteric action of the laryngeal aditus and vestibule, the elevation of the larynx and the trapdoor movement of the epiglottis.

(5) Magendie's second stage of deglutition, therefore, may be divided into the following phases:

- (a) Elevation of the larynx.
- (b) The posterior thrust of the base of the tongue.
- (c) The initial posterior-inferior movement of the epiglottis.
- (d) Further elevation of the larynx.
- (e) Final descent of the epiglottis.
- (f) Sphincteric closure of the laryngeal aditus.
- (g) Passage of a peristaltic wave down the pharyngeal constrictor muscles.
- (h) Opening of the superior end of the esophagus.
- (i) The anterior movement (return) of the base of the tongue to the resting position.
- (j) Return of the epiglottis to the upright position.
- (k) Descent of the larynx to the resting position.

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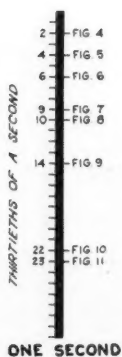


Fig. 12.—Summary of time intervals elapsing between each of the foregoing photographs taken during the swallowing cycle. The column, representing one second, is divided into thirtieths of a second.

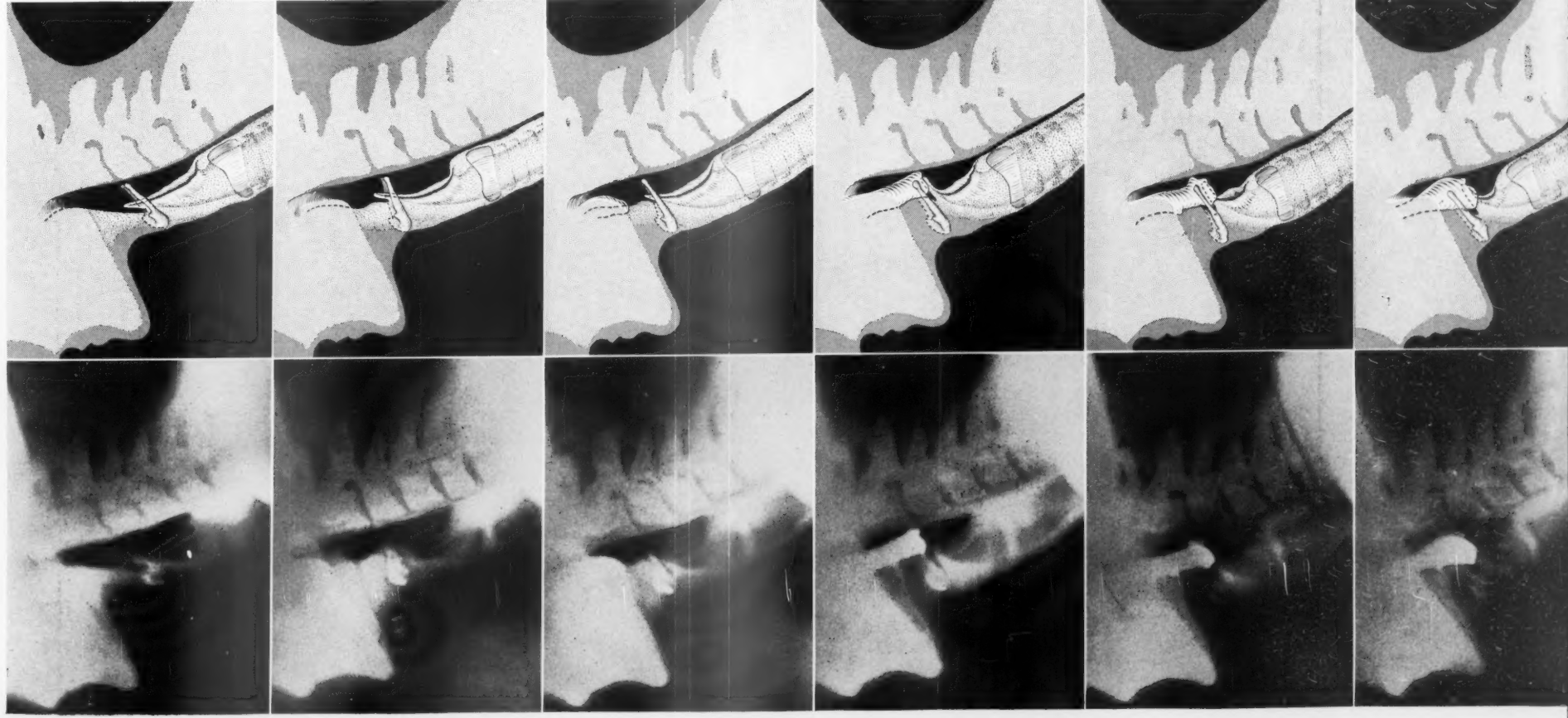


Fig. 1.—Normal subject at rest. Note the relaxed laryngeal aperture between the aryepiglottic folds, and the position of the hyoid bone in relation to the vertebrae. Observe the extent and configuration of the pharyngeal airspace for purposes of comparison with the succeeding figures. The epiglottis is upright.

Fig. 2.—Normal deglutition begins with elevation of the larynx shown here by the change in level of the hyoid bone in relation to the vertebrae, and by the rotation of the hyoid to a more horizontal position. The posterior thrust of the base of the tongue is outlined in the superior pharyngeal airspace; this motion of the tongue is responsible for the beginning (as yet slight) posterior-inferior movement of the epiglottis. Observe that the aryepiglottic folds have already begun to shorten slightly.

Fig. 3.—The bolus (thick barium) appears at the level of the fauces 27/30 second after the larynx begins its upward movement.

Fig. 4.—2/30 second later the bolus reaches the level of the valleculae. The epiglottis is moving toward the horizontal. The aryepiglottic folds are still further shortened by elevation of the arytenoid eminence as may be judged by its level in relationship to the vertebrae.

Fig. 5.—2/30 second later the leading edge of the bolus is passing the level of the hyoid bone. Note the marked shortening of the aryepiglottic folds.

Fig. 6.—2/30 second later the bolus enters the pyriform sinus by flowing around the lateral borders of the epiglottis. The epiglottis is now almost horizontal. The sphincteric action about the laryngeal aditus is increasing, although the aditus is still open.

Fig. 4.—2/30 second later the bolus reaches the level of the valleculae. The epiglottis is moving toward the horizontal. The aryepiglottic folds are still further shortened by elevation of the arytenoid eminence as may be judged by its level in relationship to the vertebrae.

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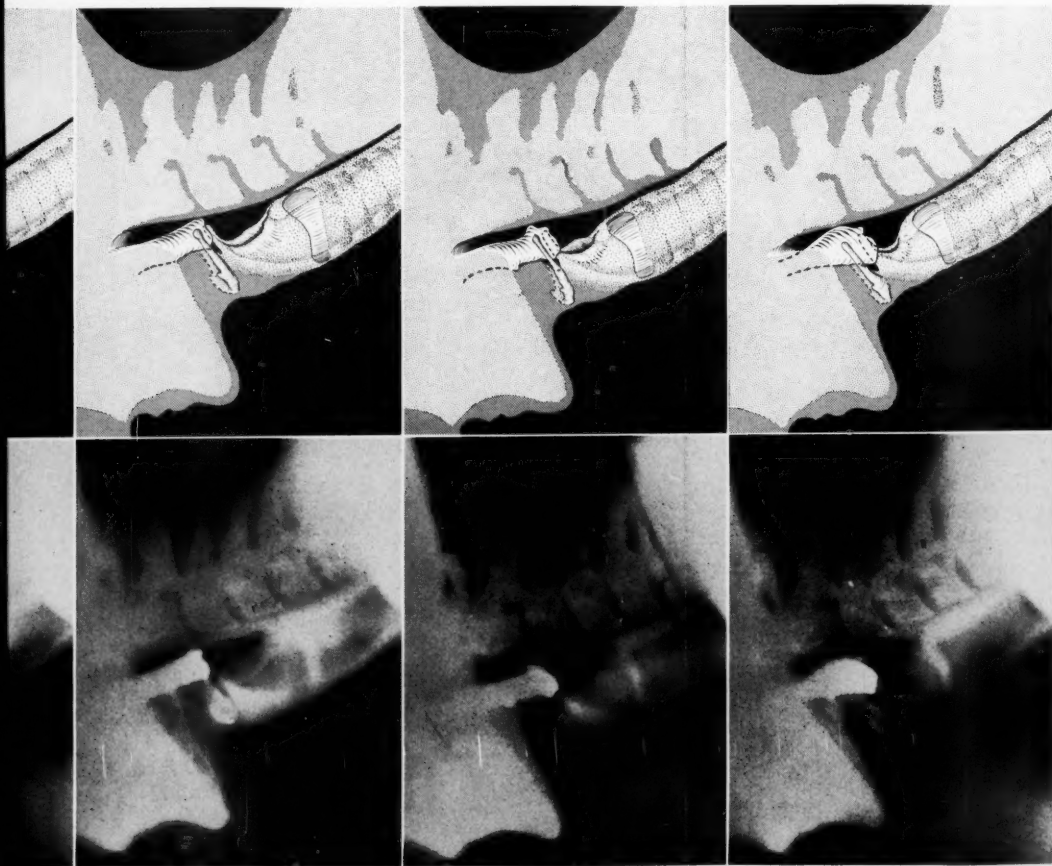


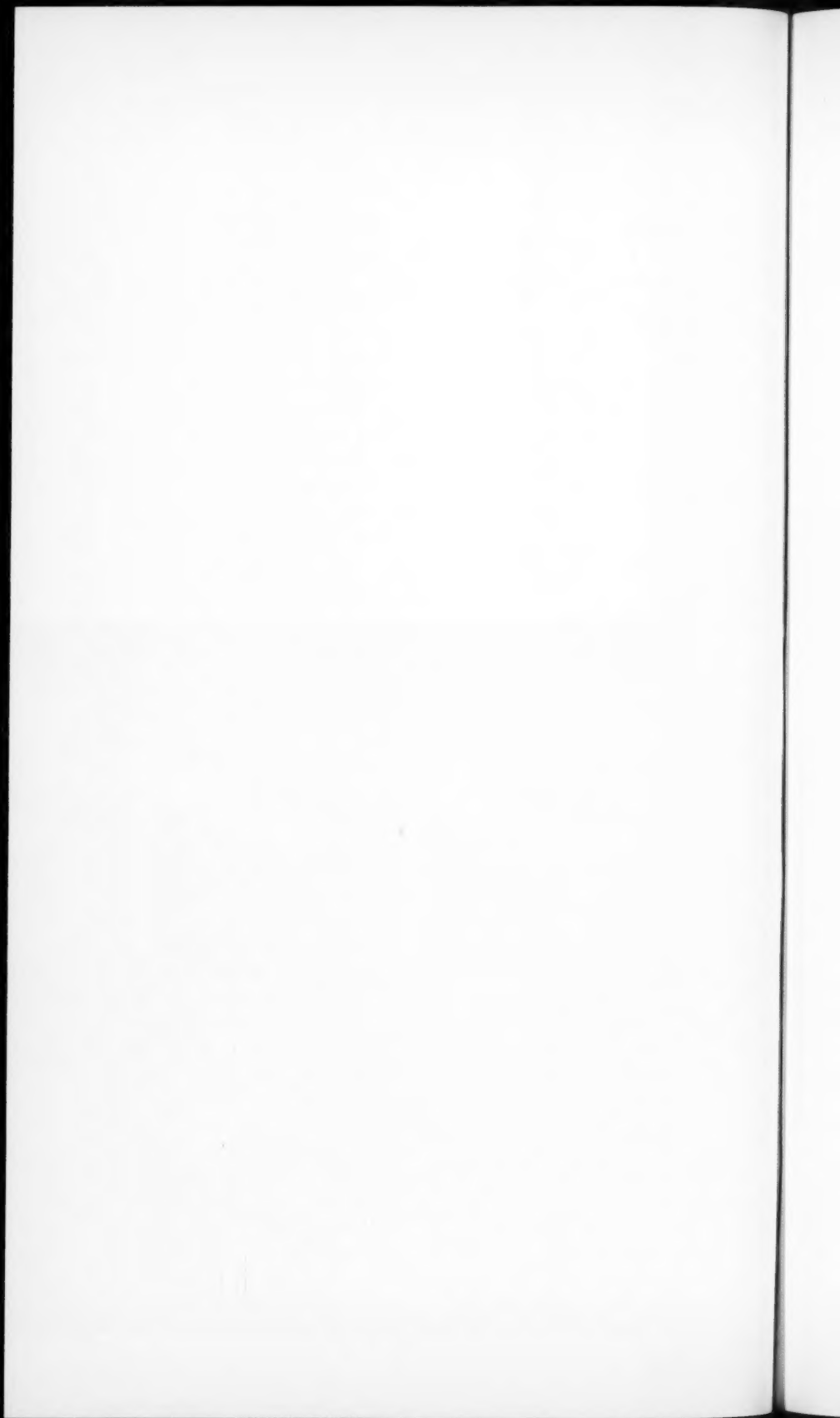
Fig. 7.—3/30 second later the leading edge of the bolus is about to enter the esophagus, having passed from the level of the fauces to the level of the cricoid in 9/30 second. The laryngeal aditus and vestibule are now completely closed. The larynx is at its maximum elevation. The epiglottis has swung down well below the horizontal.

Fig. 8.—1/30 second later the bolus has entered the esophagus. The epiglottis is now 60° below the horizontal.

Fig. 9.—4/30 second later most of the bolus has passed into the esophagus. The larynx remains closed.

Fig. 10.—8/30 second later (or 12/30 second after the leading edge of the bolus entered the esophagus) the after-end of the bolus is about to enter the esophagus. A wave of contraction has passed down the pharyngeal constrictor muscles synchronously with the descent of the bolus; this wave is here visible on the posterior pharyngeal wall just above the level of the cricoid. The larynx has opened slightly preparatory to the return of the epiglottis to its vertical position. The hyoid has dropped slightly and is starting to rotate back to its oblique position.

Fig. 11.—1/30 second later the epiglottis has returned to an almost vertical position carrying with it fragments of the bolus on its upper or ventral surface. Remnants of the bolus may be seen on either side filling the pyriform sinuses. The laryngeal aditus is open, the aryepiglottic folds are relaxed, and the arytenoid eminence has descended to its initial position. This completes the swallowing act. A second or third movement of swallowing will clear the adherent remnants of the bolus.



LXXX

PHOTOELECTRIC NYSTAGMOGRAPHY

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The standardization of vestibular examination is still far from being accomplished. Not only are there various schools of thought but almost each individual examiner employs different methods to perform the tests, the caloric one in particular. The usual cause of the numerous errors and one of the greatest hindrances to the proper standardization of the procedures can be found in the fact that the nystagmus, with all its characteristics, is a phenomenon very difficult to observe and analyze. And even the determination of the starting and stopping time of a reactive nystagmus following calorization is not simple.

In order to improve the methods of vestibular examination, the first requirement is to obviate, so far as possible, the troublesome and unreliable subjective observation. This need was recognized by the first pioneers of vestibular physiology, and the search for objective registration or graphical representation of nystagmus has been going on since their time. Many different instruments have been constructed and advocated for this purpose and the more recent ones have given excellent results in the hands of research workers in the specialized laboratory.

Surveying the efforts made to obtain graphical recordings of nystagmus, one must go back to Högyes,¹ the outstanding vestibular physiologist, who solved the problem simply by inserting a light metal rod into the eye, using the other end of this rod as a scribe over a moving paper roll. About the same time Dewar² was experimenting with electrical methods.

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Since these early days all the attempts to record nystagmus may be classified into three major groups according to the operating principles of the instruments upon which they are based.

1. Initially, mechanical devices were employed, mostly for animal experimentations. De Kleyn and Storm Van Leewen,³ Le Heux and De Kleyn,⁴ and Kuilman⁵ used various types of writing rods accomplished by means of corneal anesthesia. Ohm⁶ employed a rubber cup fitting tightly upon the eye, connected to a closed manometric system and writing apparatus.

2. A great improvement was achieved by various photographic methods, which could be applied to human subjects. Some 40 years ago, Wojatschek⁷ employed a concave mirror attached tightly to the closed eyelids, the reflected light being picked up by a cinematographic film. The same principle was later improved by the American neurologist Dodge,⁸ and similar attempts were made among others by Wiedersheim,⁹ Struyken,¹⁰ Dohlman,¹¹ Linthicum¹² and Schwartz.¹³

3. Electrical methods are the most modern development. The first real nystagmographic experimentations by electrical methods were carried out by Schott.¹⁴ Electrodes were placed into the conjunctival sac and the nystagmus recorded by use of a string galvanometer. A few years later Meyers¹⁵ and Jacobson¹⁶ described a new method quite independent of each other. By using two electrodes, one placed over each temple, a nystagmus having a horizontal direction could be recorded. Meyers used the string galvanometer of an E.K.G. apparatus for his recording. Jacobson employed for the first time a vacuum tube amplifier. Their original thought was that they were picking up the action current of the extrinsic ocular muscles.

The recent impetus toward nystagmographic studies is due to a discovery by Mowrer and his coworkers.¹⁷ They found that the action current of the lateral or medial rectus muscle is so small that even with the most sensitive electrical devices available, it could not be picked up through the thick bones of the skull. Actually the phenomena observed are based upon the persistent corneo-retinal electrical potential, the posterior part of the eyeball and the retina itself being negative, and the anterior part with the cornea being positive in relation to each other. The motion of the eye causes a shift of this potential relative to the electrodes applied at the temples.

The Mowrer principle has found many followers in different countries during the last 15 years and a series of articles have appeared concerning nystagmography based upon the corneo-retinal

potential difference. Fenn and Hirsch¹⁸ showed that the changes in corneo-retinal potential difference picked up at the electrodes is proportional to the angle of deviation of the eyes and that there is no difference in the records made with open or closed eyes. Further investigations were made by Halstead,¹⁹ Lung and Mittermaier,²⁰ Perlman and Case,²¹ Leksell,²² Baudoin and Causse.²³ Hoffman and his coworkers²⁴ compared the results and technique of photographic and electrical methods. In recent years Fabbi and Postelli,²⁵ Glorig and Mauro,²⁶ Suzuki and Yamamuro²⁷ reported their studies and Marg²⁸ gave a comprehensive survey of the literature based upon the corneo-retinal potential method.

It is not the object of this paper to evaluate the usefulness and effectiveness of the different approaches for objective recording of the involuntary eye-movements. It is evident that devices and techniques based upon the corneo-retinal standing potential are or may be real aids to vestibular diagnosis. Nevertheless, a review of the constantly increasing literature indicates that the nystagmograph is still an experimental instrument rather than an indispensable and essential diagnostic tool to be placed at the disposal of the practitioner.

The present paper describes an attempt to provide a nystagmograph that is simple to adjust and operate, that requires no electrodes, causes no discomfort to the patient, that does not interfere with the normal sequence of eye movements, and that gives a true record, on a direct pen writer, of every detail of these movements.

The instrument described here is a first model which is capable of further improvement and refinement. However, the clinical experience obtained by its use indicates that its principle of operation is entirely practicable and that it can be produced in a form which is compact and easily portable, and which can be applied to the patient's eyes almost as simply as the Frenzel glasses. The results obtained thus far show conclusively that a clear and faithful record of the nystagmus can yield interesting and important details which could not be obtained by mere observation of this phenomenon.

The principle upon which the photoelectric nystagmograph operates is as follows:

A spot of light is focused on the surface of the eyeball in such a position that it is partly on the sclera and partly on the iris. As the eyeball executes nystagmic motions, the portion of the beam that is on the sclera increases and decreases rhythmically. Since the reflection coefficient of light is greater for the white surface of the sclera than for the darker iris, the total amount of reflected light

increases and decreases proportionally with the eye motions. A photocell placed immediately in front of the eye picks up this varying reflected light and generates a correspondingly varying electric current, which is amplified electrically and made to actuate a direct pen writing recorder.

The shape of the light spot and its position on the eyeball is illustrated in Figure 1. This figure illustrates the case for left ear cold calorization. For right ear calorization the light spot would be focused so as to protrude beyond the opposite margin of the iris. The spot is in the form of a narrow rectangle approximately 1 mm by 10 mm. This form was chosen because it gives a linear relation between the magnitude of the eye displacement and the corresponding change of reflected light intensity. The position of the spot is such that it crosses the borderline between sclera and iris both when the subject is looking straight ahead (position a) and when he is looking to his right (position b). In either position, the small nystagmic motions will expose more of the scleral surface to the light in the rightward displacements and less of this surface in the leftward. The fact that in position "b" much more of the spot is constantly on the sclera is of no consequence since the apparatus is so constructed that the recording pen motion is determined only by changes in reflected light, that is, by the extent of the nystagmic movement of the eye. It is obvious that the light spot must be properly oriented on the eyeball. If it is placed too far to the subject's left, it will be entirely on the sclera when the eye moves to position "b." The nystagmic movement will then produce no changes of light intensity and no record will be obtained. On the other hand, if the spot is too far to the right it will lie entirely in the iris in position "a" and again no change of light intensity will result.

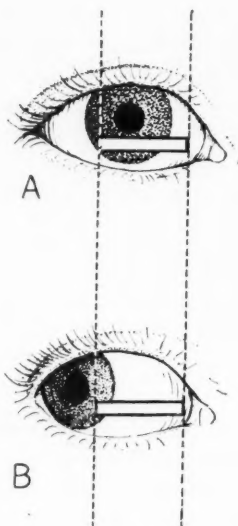


Fig. 1.—The light spot: its shape and position.

For the observation of vertical nystagmus, the light spot is positioned vertically on the eyeball so as to protrude into the sclera below the margin of the iris.

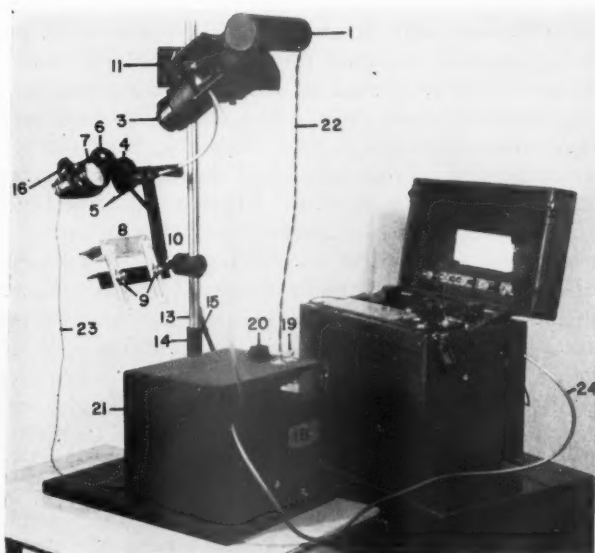


Fig. 2.—The Apparatus.

A fundamental difficulty which had to be overcome in the design of the apparatus arose from the fact that, while the eye of the subject must necessarily be illuminated in order to produce a photoelectric effect, the subject must not be presented with a light spot or any other image upon which he can fixate, since fixation and undue light tend to minimize or suppress nystagmus. The difficulty was met by using invisible infrared light for illuminating the eyeball and by making use of the principle of the Frenzel glasses, that is, by placing a very thick (16 diopter) lens before the subject's eyes, the optical system of the illuminator being so designed that a properly defined light spot, as described above, can be cast on the eyeball through this thick lens. It would, of course, have been possible to use ultraviolet instead of infrared illumination. The latter was chosen because at equal intensities ultraviolet is potentially more injurious to the eye and because good sources of infrared (incandescent light filaments) are readily available and very sensitive infrared photoelectric cells have been developed.

Details of construction are shown in Figure 2. The light source, a straight filament incandescent lamp drawing 3.5 amperes at 6 volts, is contained in the lamp housing (1). This housing may be

rotated in its tubular support to orient the light spot on the eyeball vertically or horizontally or in any desired position. It may also be moved in an axial direction for focusing the light spot. Light passes from this source through an optically flat infrared light filter. (Corning glass color filter No. 7-69). This filter slides into a slot in the lens housing and can be readily removed from the light path with a small knob. From this filter the light passes through a high grade compound lens (3), of F-2 aperture and seven-inch focal length, and through the 16-diopter simple lens (4) near its center. This optical system is capable of giving a very sharp image of the filament on the eyeball. Actually however, a "soft" focus is used with an image width of 1 to 2 mm since this tends to average out slight local inequalities of the mottled iris coloration as well as local variations of sclera color due to blood vessels, etc. The 16-diopter lens (4) is actually in the form of a half-lens as shown, the lower half having been cut away to permit the photocell (5) (RCA No. 1P41) to be brought close to the eye and to avoid the loss of reflected light intensity that would be caused by passage through the lens. A goggle frame (6) holds this half lens and the photocell and also a whole lens (7) of 16-diopter strength before the opposite eye. A chin rest (8), together with the goggle frame, serves to position the subject's head relative to the illuminator and to hold it in the so-called "optimal position" of the horizontal semicircular canals following calorization. The chin rest is adjustable by means of the thumbscrews (9). The interpupillary distance of the goggle is adjustable by means of a friction joint and its position relative to the chin rest is adjustable by means of thumbnuts (10). The whole illuminator may be adjusted in vertical angle and distance relative to the goggle by loosening a thumbnut (not shown) and moving it in its support (11). It may be adjusted in the horizontal angle by swiveling in the support. Finally, the whole illuminator and goggle assembly may be raised or lowered by moving the support rod (13) in the stand (14), it being fixed in the desired position by the hand lever (15).

A small shielded light (16) illuminates the subject's right eye without glare to provide good observation of this eye, if desired, as a supplement to the recordings. Such observations may be useful, for example, to detect the presence of rotatory nystagmus or other unusual qualitative features. This light is controlled by a switch and obtains its current from a small step-down transformer operated from the 110 volt a.c. power supply. The light source (1) must be operated on direct current since a.c. operation causes variations of light intensity at 120 cycles per second which interfere seriously with

the recordings. The direct current is furnished by a rectifier and filter housed in the case (18). It is controlled by the switch (19) and a rheostat (20). The case (21) contains a small 90 volt battery for the photocell power supply. Cords (22 and 23) connect respectively to the photocell illuminator and the small lamp (16). The shielded cable (24) carries the photocell output current to the electronic amplifier and pen writer assembly which, in the set-up shown in Figure 2, is a standard Sanborn portable electrocardiograph. While this instrument gives excellent records it is not ideally suited, in frequency response characteristics, to the present use, and it would be possible to produce an amplifier and pen writer of simpler design which would be more practical for this particular purpose.

The making of the record is carried out as follows:

The subject is seated before the apparatus and all necessary adjustments are made according to his height, chin to eye distance, and interpupillary distance, with the devices described above, so as to give a proper and comfortable posture. The illuminator switch (19) is turned on, and with the infrared filter removed, the light intensity is adjusted by the rheostat (20) to give an easily visible but soft yellow light spot on the eyeball. This is properly positioned and focused by means of the various adjustments provided and locked into position by tightening the thumbnuts. The subject is then removed, the calorization is performed, the infrared filter is placed in position, and the illuminator intensity brought to maximum by means of the rheostat. At the proper moment the subject is returned to his previous position and a record is made.

With the equipment shown it is possible to obtain a motion amplification of ten or more, that is, with a motion of the eyeball surface of one millimeter the pen excursions may easily be adjusted to ten millimeters. Thus even a nystagmus which is too small to be observed directly may still be clearly recorded.

Records of various types of nystagmic motions are shown and described in Figure 3. It will be noted that the various records show pronounced quantitative and qualitative differences. For example, records 1, 2, 3 and 4 differ considerably in frequency and amplitude and in the relative duration of the slow and fast components. Also while in 1 and 4 the slow component is predominantly monophasic, it is multiphasic in 2 and 3. These four records all show the saw-tooth pattern which is characteristic of reactive caloric nystagmus, and also of other types of vestibular nystagmus. This is in marked contrast to the undulatory character of the optic nystagmus 5 and the square wave pattern 6. Some of these variations in detail, which

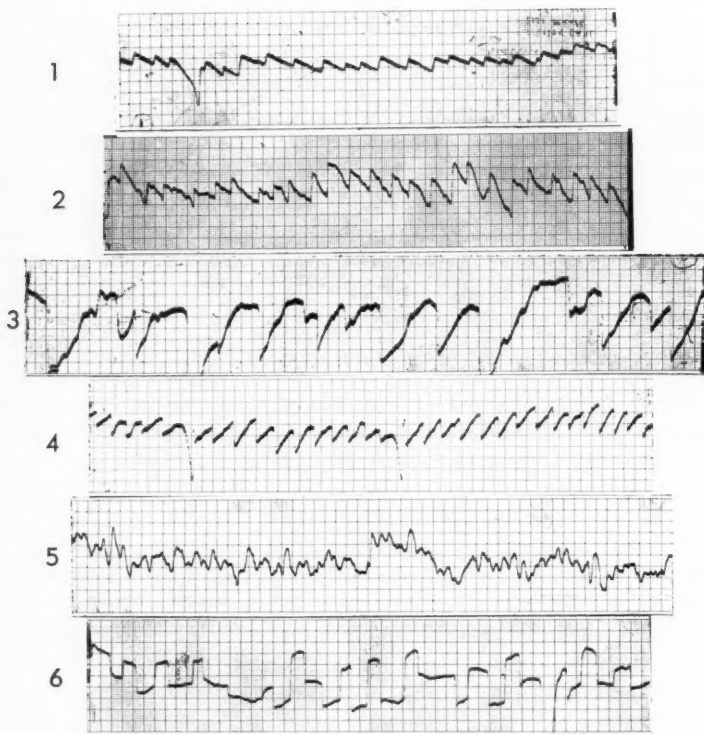


Fig. 3.

1. Reactive caloric nystagmus. Small amplitude. Frequency slightly above the average.²⁹
2. Reactive caloric nystagmus. Medium amplitude. Frequency slightly above the average.
3. Reactive Caloric nystagmus. Coarse eye motions. Large amplitude. Reduced frequency.
4. Spontaneous vestibular nystagmus of high frequency.
5. Spontaneous optic nystagmus.
6. Nystagmus-like rythmical eye motion. Appeared at the end of a normal caloric response. This phenomenon may be misinterpreted and considered as the end-phase of the reactive nystagmus.

are quite obvious in the records but which would be difficult or impossible to pick out by visual observation, may well be of considerable diagnostic significance. Their proper interpretation will however, require the study of many more records and correlation with other clinical findings.

SUMMARY

In order to standardize the technique of vestibular examination, by means of the phenomenon of nystagmus, it is essential to eliminate subjective observation. Methods for obtaining objective recordings of nystagmus have been known for the last 70 years.

Early instrumentation of the mechanical type was suitable for experiments on animals only, but the later optical devices could be used on human subjects as well. Of the more recent electrical instruments, the most successful are based on the discovery of the permanent corneo-retinal potential of the eye. While these have yielded clear and reliable recordings, they are still essentially experimental research instruments rather than diagnostic tools for clinical vestibular examination.

An apparatus is described which utilizes a photoelectric cell to record variations in the reflection of an infrared light beam focused on the eyeball. Faithful records are obtained showing the characteristics of various types of nystagmus and nystagmus-like motions. The apparatus is simple to use, no electrodes are employed, and there is no discomfort to the patient.

Examples are shown of various records, both spontaneous nystagmus and nystagmus by calorization. The characteristic significance of the various types is pointed out.

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VESTIBULAR ROTATORY AND OPTOKINETIC
REACTIONS IN THE PIGEON

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These experiments were inspired by a communication by Mowrer.¹ He devised a method to record the head-nystagmus of the pigeon, which was used in these experiments. During and after rotation various combinations of vestibular and optokinetic nystagmus are possible. The results described by Mowrer could be completely confirmed. These experiments were enlarged upon by an examination of the reactions of decerebrated pigeons and of pigeons that had one labyrinth removed or section performed on one horizontal semicircular canal. The optokinetic nystagmus was also examined by means of a cylinder as devised by Visser.²

APPARATUS

With the rotatory and the post-rotatory reaction of the pigeon the head-nystagmus is strongly prevalent. The pigeon also has an ocular nystagmus, which is much less pronounced. Apart from this, the head-nystagmus of the pigeon shows exactly the same characteristics as e.g. the ocular nystagmus of man, the same qualities can be discerned in it. It seemed important to us also to examine the optokinetic nystagmus by itself. For this purpose a rotating cylinder with optic contrast was used, as was already done by Visser on the pigeon. Fig. 1 shows the apparatus, which was used in our experiments. The pigeon is tied up in the pigeon-holder which can be turned to the right or to the left by means of a motor. We always used a strong stimulus for the experiments, viz. an angular velocity of 180° per second. The starting of the rotation as well as the stopping is done very suddenly. A second motor can rotate the optokinetic cylinder to the right as well as to the left. During the experiment the cylinder is also covered by a lid with the same black and white stripes. In the center of this lid is a small opening, over

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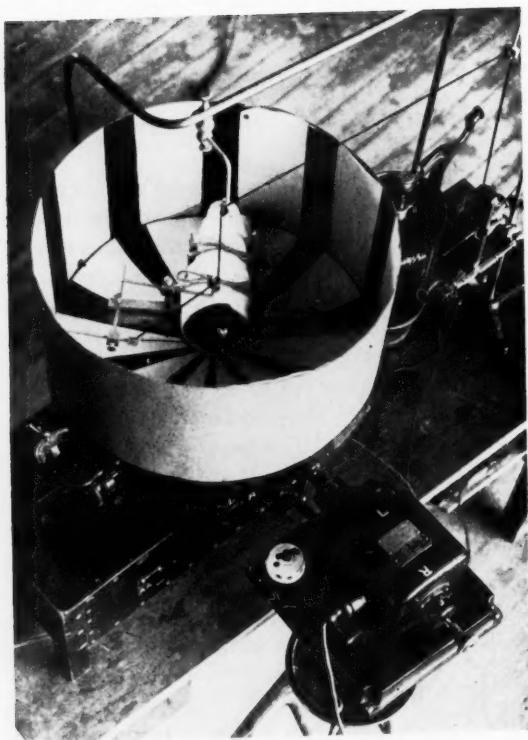


Fig. 1.

which a ground glass electric bulb is fastened. In this way a fine diffuse lighting of the cylinder was obtained. The room was blacked out. By turning the lamp on or off vision could be permitted or excluded at will during the rotation and immediately after it. A silk thread is tied through a tiny hole in the tip of the pigeon's bill. The movements of the head are recorded on a kymograph. The thread runs from the bird's bill through a glass eyelet, which is always to the right of the bird's head, and then to the small pulley exactly in the axis of the rotation. During the rotation the thread between the first and second pulley is twisted, but the change in length is so slight that it does not influence the record. On the curve we see only the movements of the head to the right and the left. The curve is written by a very light lever-marker which moves strictly vertically. The thread runs over a third pulley to this marker. It is clear that a movement of the head to the right gives a downward movement of the marker and a movement of the head to the left gives an upward movement on the kymograph. When there is a quick nystagmus it is often difficult to distinguish the slow from the quick phase through the turning of the kymographion. On the contrary this is very easy when the nystagmus is slow. Several of the following curves show this very clearly. On the kymograph the time in seconds and the rotations of the pigeon and also of the optokinetic cylinder are recorded. Completely in agreement with Mowrer, we succeeded in obtaining very fine records of the head-nystagmus of the pigeon.

VESTIBULAR ROTATORY REACTIONS

In order to get experience concerning course of the vestibular rotatory reactions of the normal pigeon, a large number of pigeons was examined in the dark. Thus optokinetic reactions and disturbances due to fixation of a point in the surroundings, if any, were completely eliminated.

A striking difference of intensity and duration of the rotatory reactions of the various pigeons exists. Extreme differences are found. A few instances: Pigeon 619: rotation to the left in the dark results in a rotatory nystagmus of 25 seconds and a post-rotatory nystagmus of 27 seconds. Pigeon 599 has a rotatory nystagmus of 11 seconds and a post-rotatory nystagmus of three seconds. The same pigeon may also give considerable difference in the reactions at various points of time, e.g.: Pigeon 609: rotation to the right in the dark, rotatory nystagmus 20 seconds, post-rotatory nystagmus 12 seconds. Three days later: rotatory nystagmus 18 seconds, post-rotatory nystagmus three seconds.

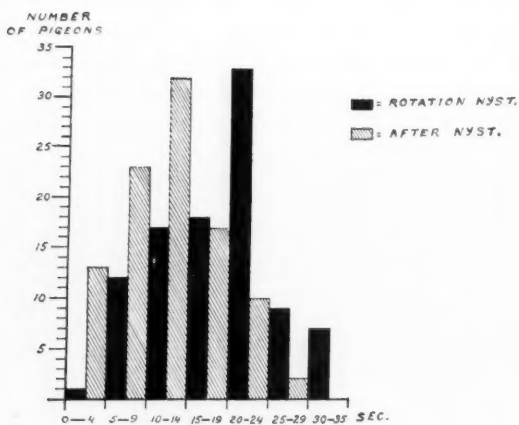


Fig. 2.—Duration of the rotation and afterreaction in 92 pigeons.

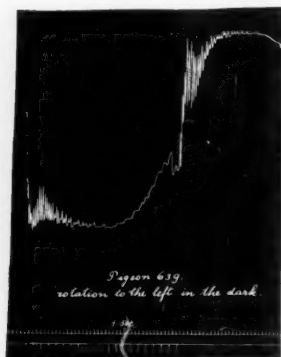


Fig. 3.—Reaction and afterreaction in the dark. Pure vestibular nystagmus. Rotation to left.

The differences in the duration of the nystagmus are closely related to the condition of the pigeon, e.g. saturation of the crop, emotion. It often makes a great difference whether the pigeon has been caught after a chase or has been quietly sitting in a pen. In the latter case the graph was flatter and the nystagmus of shorter duration, but also much less disturbed by voluntary movements. As a rule young pigeons give better records than older ones. For these reasons we saw to it that the various experiments were performed under conditions of the most possible similarity.

Fig. 2 is the diagram of the duration of the nystagmus obtained from curves of 97 normal pigeons. It appears that the duration of the rotatory nystagmus most frequently lies at over 20 seconds, the post-rotatory nystagmus is of shorter duration. On the diagram of Fig. 2 the great dispersion in the duration of the nystagmus of various pigeons immediately catches the eye.

Similar results as on the diagram of Fig. 2 are also found with man. This is even a great difficulty for the clinic. In a certain case no great value can be attached to the duration of the nystagmus. Much more valuable are obvious differences between the results after rotation to the right and to the left at a given moment.

Fig. 3 is a typical example of a record of a normal pigeon turned to the left in the dark. Below the nystagmus curve are two lines; the top one indicates the time in seconds, the bottom one shows a top when the pigeon has been rotated once. This is done by means of an electric contact. The velocity of the rotation always amounted to one rotation in two seconds. Here the movement was stopped after 20 rotations. This is necessary as the rotatory reaction sometimes, takes more than 20 seconds; for this reason it is better to rotate with a uniform velocity for a longer time than the 20 seconds usual in the clinic. The error of examining the post-rotatory reaction at a point of time when the rotatory reaction has not yet stopped is often committed. This error can be evaded by exclusively examining the post-rotatory reaction, the velocity of the rotation being raised very slowly so that a rotatory reaction does not occur.

We see on Fig. 3 that at the start of the rotation the pigeon's head makes a wide movement to the left, the direction opposite the rotation. This is immediately succeeded by the nystagmus with the swift movement in the direction of the rotation and a slow movement opposite to it. The nystagmus movements reach their maximum after a few seconds and then gradually decrease. The head returns to the central line; when the rotation is stopped the head

makes a movement to the left and an evident nystagmus now appears, having its swift phase to the right. This is the post-rotatory reaction.

COMBINATION OF VESTIBULAR AND OPTOKINETIC NYSTAGMUS

Fig. 4 is a typical example, chosen from 52 records, of a rotatory reaction completely occurring in the light during and after turning to the left. The beginning is as in Fig. 3, once more a considerable deviation of the head is seen in the direction opposite the rotation, and a nystagmus in the direction of the rotation. The deviations, however, are immediately very considerable and continue throughout the rotation. The initial nystagmus has its origin in a combination of vestibular and optokinetic stimuli. During the latter part of the rotation the nystagmus is apparently completely kept up by optic stimuli as was also demonstrated by Mowrer. He speaks of "pursuit movements," thus assuming the fixation to play the only part in this phenomenon. This is certainly not correct. With this apparatus the subcortical optokinetic nystagmus plays the most important part. This will be discussed later.

After the rotation is stopped the head returns to the central line. This, therefore, is a movement opposite the direction of the rotation and there is a post-rotatory nystagmus in the direction opposite to that of the rotatory nystagmus, of strikingly short duration. This can be partly explained by the fact that after the rotation the animal can fix, thus suppressing the nystagmus. But there is another important difference in the situation in the course of the post-rotatory reactions of Fig. 3 and 4. In Fig. 3 the head is at rest when the rotation is stopped. In Fig. 4 a strong nystagmus exists when the rotation is stopped; this is of great importance for the post-rotatory reaction. This fact was already called to attention by Bárány in man. When the head was at rest during the rotation, the post rotatory nystagmus was of much longer duration than when the head followed the rotation with jerking movements, a condition, therefore, as in Fig. 4. This device is also used by dancers to suppress post-rotatory dizziness. Bárány in explaining this said that the cristae are subjected to stimuli again and again from opposite directions, so that one cancels the other. When one swiftly turns one's head 180° nystagmus does not occur. The rotatory reaction is immediately neutralized by the post-rotatory reaction.

According to Ewald³ the post-rotatory reaction is completely absent under the circumstances of Fig. 4. This is decidedly not correct, although the post-rotatory reaction is usually very short.

Fig. 5 is the typical example of a record chosen from 24 curves, of a pigeon rotated in the dark to the left, the light being switched on immediately after the rotation. In this record a nystagmus begins immediately when the rotation is started. The quick phase is in the same direction as the rotation, as always the slow phase precedes the quick one. After that the strong head-deviation begins with the direction opposite the rotation. The nystagmus is interrupted by a few voluntary movements. During the latter half of the rotation the head is completely at rest. The post-rotatory reaction shows a very strong deviation in the direction of the rotation and a nystagmus with the quick phase to the right of a much shorter duration than in Fig. 3 but still longer than in Fig. 4. The animal can fix now, thus suppressing the nystagmus, but the vestibular stimulus is stronger than in Fig. 4, the head being at rest when the rotation was stopped, and the cristae thus not being subjected to opposite stimuli.

Fig. 6 is chosen from 25 records as a typical example of a pigeon rotated to the right in the light, the post-rotatory reaction taking place in the dark. During the rotation the result is exactly the same as in Fig. 4, a strong nystagmus immediately sets in, the quick phase being in the direction of the rotation and again clearly starting with a slow phase. Gradually a strong duration of the head develops. Just as in Fig. 4, the head nystagmus remains very considerable during the rotation.

As to the post-rotatory reaction, Fig. 6 displays a situation quite different from Fig. 4. After the rotation we see a strong deviation of the head in the direction of the rotation with four nystagmus strokes, the quick phase being opposite the rotatory nystagmus, so to the left. While the head returns to the central line we can later see a small nystagmus, during a number of seconds, opposite to the preceding one, viz. having its quick phase to the right. In this case we deal with a very important phenomenon also described by Mowrer: the post-rotatory reaction contains two components, the vestibular and the optokinetic post-rotatory nystagmus. For there is also an optokinetic post-rotatory nystagmus that has the same direction as the optokinetic rotatory nystagmus. This contrasts, therefore, with the vestibular reactions, where it is principally so important that the rotatory nystagmus and the post-rotatory nystagmus always run opposite to each other. So during the rotation the vestibular and the optokinetic nystagmus reinforce each other, as is so clearly visible in Figs. 4 and 6. After the rotation, on the other hand, they counteract each other.

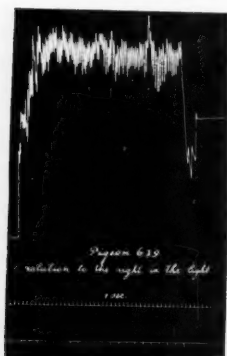


Fig. 4.—Reaction and afterreaction in the light combination of vestibular and optic reactions. Rotation to the right.

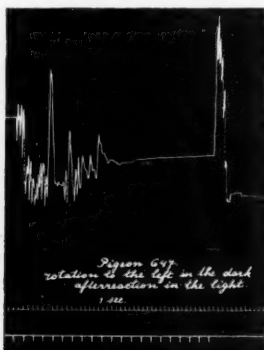


Fig. 5.—Reaction in the dark, afterreaction in the light. Rotation to the left.

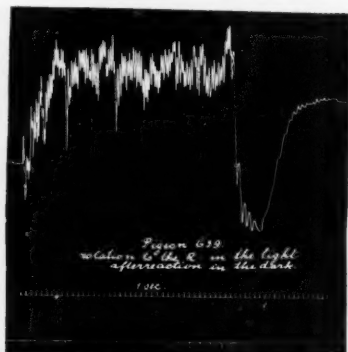


Fig. 6.—Reaction in the light, afterreaction in the dark. Rotation to the right.

With the post-rotatory reaction of Fig. 6 it is evident that at first the vestibular reaction prevails; after that there is a pause and finally the optokinetic post-rotatory reaction takes place.

We examined the same four possibilities that were indicated by Mowrer: 1. rotatory and post-rotatory reaction in the dark; 2. rotatory and post-rotatory reaction in the light; 3. rotatory reaction in the dark, post-rotatory reaction in the light; 4. rotatory reaction in the light, post-rotatory reaction in the dark.

Owing to the combination of vestibular and optokinetic reactions and the possibility or impossibility of fixation, completely different results are obtained. Mowrer's findings were completely affirmed and we agree with his explanation in almost everything. A fifth possibility can be added to these four, viz. rotating the visual field more quickly than the animal. Its result can be explained easily in relation with the preceding ones.

Fig. 7 is a typical example. The pigeon is rotated to the right, but, as appears from the line above the second's line, the cylinder is rotated still more quickly to the right. The rotation is performed in the light, the post-rotatory reaction takes place in the dark. The situation during the rotation in Fig. 7 is quite different from the one in Fig. 6. In Fig. 6 the visual field of the pigeon turns to the left, in Fig. 7 the visual field moves to the right, also with respect to the pigeon. Consequently during the rotation in Fig. 7 the vestibular and optokinetic stimuli have opposite directions.

Fig. 7 at first shows an evident vestibular reaction. There is a deviation of the head opposite the direction of the rotation and a nystagmus in the direction of the rotation. The head returns to the central line and an optokinetic nystagmus to the left sets in, the vestibular stimulus is defeated, as a matter of fact it is already partly spent. With the post-rotatory reaction we have to do with a vestibular and an optokinetic post-rotatory nystagmus, in this case, however, they cooperate. They are both directed to the left and so a very strong nystagmus to the left results.

From Fig. 7 it appears that if this method is used it takes some time for the vestibular nystagmus to change into the optokinetic one. The reverse was never found. We performed experiments in which the cylinder was rotated first and 20 seconds later the pigeon. Immediately the optokinetic nystagmus changed into a vestibular nystagmus. In our experiments the optokinetic nystagmus gave way to the vestibular one. It should be taken into account, however, that we always used very strong stimuli, viz. an angular velocity of c .

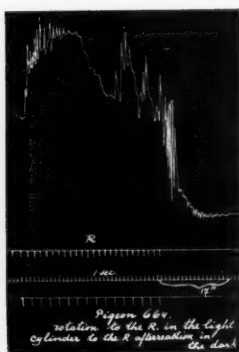


Fig. 7.—Rotation of the pigeon to the right in the light. Optokinetic cylinder turning to the right. Afterreaction in the dark.

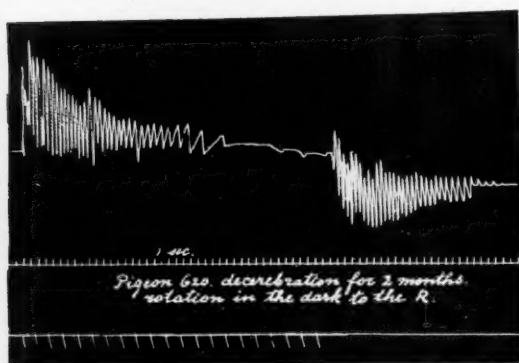


Fig. 8.—Decerebrated pigeon. Rotation to the right. Pure vestibular reactions. Rotating- and afterreaction in the dark.

180° per second. With weaker vestibular stimuli quite different results are naturally to be expected.

All this is also of the greatest importance for the clinic. It is obvious that, if one wants to get an impression of the course of vestibular rotatory reactions in man, the occurrence of optokinetic reactions and the possibility of fixation must be carefully excluded. In practice this rule is repeatedly broken.

EXAMINATION OF DECEREBRATED PIGEONS

In order to examine the influence on the cerebral cortex the same experiments were performed on decerebrated pigeons. A great number of these animals died during the examination. Eight of them were kept alive for a longer time; on these a complete examination was performed. As a rule very fine records were obtained from decerebrated pigeons; disturbing movements of the head did not occur. Numerous investigations have long since demonstrated that after removal of the cerebrum the vestibular reactions remain completely unimpaired. With the pigeon they are much stronger and more regular in their course.

Fig. 8 is the record of a decerebrated pigeon during and after rotation to the right, both the rotatory and the post-rotatory reaction taking place in the dark. If we compare this record to the one in Fig. 3 one is struck by various points. The curve is much more regular, both the rotatory and the post-rotatory reactions are very pronounced and of a long duration, 27 and 24 seconds respectively. In ten of the 19 satisfactory curves in different decerebrated pigeons values were found of 30 or more seconds even. Both rotatory and post-rotatory reaction are of a longer average duration than with the normal pigeon. An important fact is that the individual differences are much smaller. Moreover, examination of the same decerebrated animal on different days gave nearly the same results. From this it is evident that the presence of the cerebrum has a very unfavorable influence on the result of the vestibular examination. This also explains why the interpretation of the results of the vestibular examination of man gives so many difficulties in the clinic.

In Fig. 8 we are also struck by a certain conformity between the rotatory and the post-rotatory reaction. When the cortical influences have fallen out the reflex movements are undisturbed and are determined by their own laws. Much better than with normal pigeons, the same vestibular stimuli now produce one and the same reaction.

There is another difference between the curves of Figs. 3 and 8. Whereas the nystagmus on Fig. 8 is much more pronounced, the deviation of the head, as opposed to Fig. 3, is only very slight. From experiences also gained from normal pigeons, it appears that this too reinforces the head-nystagmus. When the deviations of the head is very marked the head-nystagmus usually decreases.

Fig. 9 is analogous to Fig. 4. Both the rotatory and the post-rotatory reaction take place in the light. The decerebrated pigeon is turned to the right. We see a very marked nystagmus during the rotation because the vestibular and the optokinetic nystagmus cooperate. This proves, therefore, that the optokinetic nystagmus of the pigeon can still occur after removal of the cerebrum. Fixation plays no part in this; there can be no question of "pursuit movements." In this case we deal, therefore, with a subcortical form of an optokinetic nystagmus. The post-rotatory reaction is of a much shorter duration than in Fig. 8. This was already sufficiently explained with the normal pigeon; the head-movements at stopping hinder the post-rotatory reaction. The post-rotatory reaction is longer, however, than with the normal pigeon of Fig. 4. This must be explained by the fact that fixation is no longer possible for the decerebrated pigeon.

In Fig. 10 the decerebrated pigeon is rotated to the right in the dark whereas the post-rotatory reaction takes place in the light. Very striking is the complete similarity of the curve obtained during the rotation to the one of Fig. 8 of the same decerebrated pigeon. The meaning of this fact has already been pointed out. The post-rotatory reaction is seen to be much stronger than with the normal pigeon of Fig. 5. This may be explained by the absence of fixation; still, the post-rotatory reaction is considerably shorter than the one of Fig. 8, in which the post-rotatory reaction took place in the dark. So another factor, not yet discussed until now, must play a part. Ter Braak⁴ demonstrated that optokinetic nystagmus can already set in when a small image moves a short distance along the retina. Owing to the movement of the head, due to the vestibular post-rotatory nystagmus, it will, therefore, also be possible for a subcortical optokinetic nystagmus to set in, which may be responsible for the shortened post-rotatory reaction.

Fig. 11 can be compared with Fig. 6 of the normal pigeon. The rotation to the right of the decerebrated pigeon is performed in the light; the post-rotatory reaction takes place in the dark. The deviation of the head is smaller with the decerebrated pigeon than with the normal one. There is a very marked nystagmus during the ro-

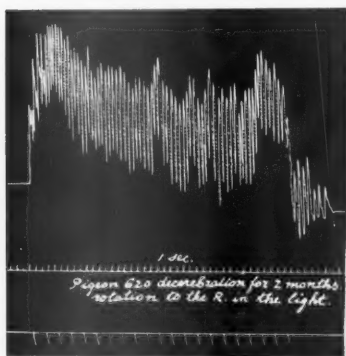


Fig. 9.—Decerebrated pigeon. Rotation and afterreaction in the light. Rotation to the right.

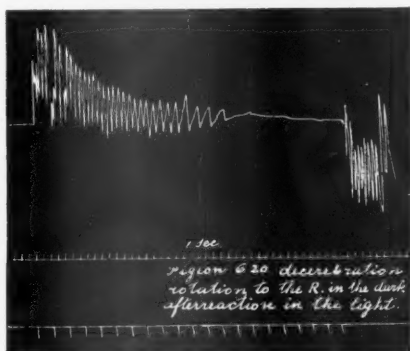


Fig. 10.—Decerebrated pigeon. Rotation in the dark, afterreaction in the light. Rotation to the right.

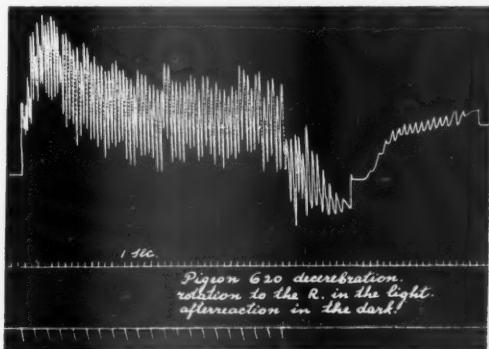


Fig. 11.—Decerebrated pigeon. Rotation in the light, afterreaction in the dark. Rotation to the right.

tation due to cooperation of the vestibular nystagmus and the optokinetic one. The post-rotatory reaction displays the same interesting features. First a vestibular post-rotatory nystagmus sets in lasting about ten seconds and after a short latent period of a few seconds, in which the two post-rotatory reactions are balanced, the optokinetic nystagmus prevails, in this case lasting about 16 seconds. So the course of things is again such that at first the vestibular stimulus prevails and is then gradually cancelled. Then a fairly prolonged optokinetic post-rotatory reaction follows. This reaction lasts until 26 seconds after the end of the rotation, a duration never found with an optokinetic post-rotatory reaction not accompanied by a vestibular stimulus. One gets the impression that during the vestibular post-rotatory reaction the optokinetic post-rotatory reaction remains present and is discharged when the crista has stopped. It looks as if only the point of time is shifted.

From the experiments described here, it appears that the reactions of the decerebrated pigeon are not essentially different from those of the normal pigeon. Both vestibular and optokinetic nystagmus are present; there is always an addition, the two forms reinforce or detract from each other according to their directions. The vestibular nystagmus as well as the optokinetic one, therefore, have a subcortical origin. This only applies to a part of the optokinetic nystagmus, however. It is striking that, whereas the vestibular nystagmus is more marked with the decerebrated pigeon, the optokinetic nystagmus has decreased. This can be explained by a partial falling out of the optokinetic nystagmus as will be described below.

These results are also important for the clinic. It is evident that with our patients the presence of the cerebral cortex with its ever changing influence is very disturbing for a correct judgment of the course of the vestibular reactions.

OPTOKINETIC NYSTAGMUS

For a long time it has attracted attention that simultaneous existence of vestibular and optokinetic nystagmus results in addition and not in superposition. The two components cannot be distinguished any more. Special attention was drawn to this fact by Ohm.⁵ He assumes that ultimately the vestibular and the optokinetic nystagmus are generated in a common centre in the brain stem. Ohm and also Bárány⁶ held the opinion that the reflex arc of the optokinetic nystagmus runs over the cerebral cortex, the optic cerebral cortex being of particular importance. Bartels,⁷ on the other hand, assumed the reflex arc to have a subcortical course. This is conclusively proved

by the fact that it is possible to generate a fine optokinetic nystagmus in a decerebrated animal. Rademaker and de Kleyn⁸ performed an ingeniously thought out experiment. They evoked an optokinetic nystagmus in the dog by having it look at a rotating disk with living rabbits on it. Many dogs show a most vivid interest in that. A large position of the brain could be removed without disappearance of the nystagmus, but after removal of the optic centres no more optokinetic nystagmus was present. So there is much controversy in the different opinions.

Ter Braak's experiments have shed more light in this point. He distinguishes two kinds of optokinetic nystagmus: 1. the "Stier-Nystagmus" (stare-nystagmus) or subcortical form, caused by the shifting of optic contracts on the retina; 2. the "Schau-Nystagmus" (look-nystagmus) or cortical form. With the latter a moving contrast is followed. Attention as well as fixation are of the greatest importance. In man and in the higher animals these two play a much more important part than in the lower animals. Rademaker en de Kleyn, therefore, examined the cortical form only with their experiment with the dogs, which are interested in the rotating rabbits.

When examining man and the normal test animal both forms can always be engendered, even in animals like rabbits and pigeons some fixation is always present. The only method to examine one form with certainty, viz. the subcortical one, in the test animal is decerebration. Still, with de Kleyn one can in the clinic distinguish to a certain degree. The cortical nystagmus can be examined by making the patient look at moving pictures he is interested in, and the subcortical form by rotating a cylinder with black and white stripes around the patient.

In the neurologic clinic the optokinetic nystagmus has acquired more importance of late years, among other things for patients suffering from hemianopsia.

With pigeons it is very difficult to distinguish between the cortical and the subcortical optokinetic nystagmus. An experiment like the one used by deKleyn and Rademaker on dogs is not possible. Very probably the subcortical form is the more important one in the pigeon. We think that under certain circumstances the cortical form has also played a certain part.

It is very easy to bring about an optokinetic nystagmus in a pigeon by means of the rotating cylinder with optic contrasts. Fig. 12 is a typical example, chosen from 63 curves of different normal pigeons. The pigeon stands still, the field of vision moves to the

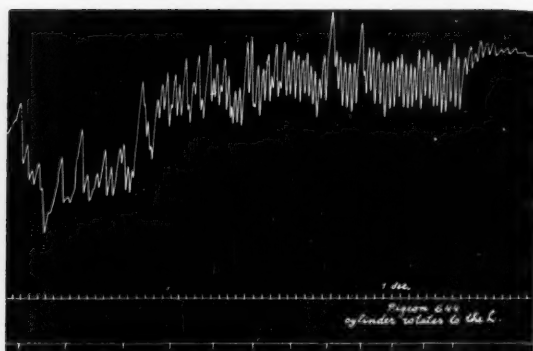


Fig. 12.—Optokinetic nystagmus in a normal pigeon.

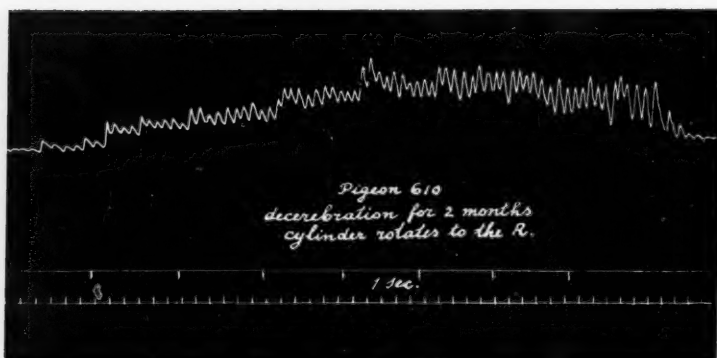


Fig. 13.—Optokinetic nystagmus in a decerebrated pigeon.

left, the nystagmus starts with a slow movement in the direction of the rotation, followed by a swift movement in the opposite direction, the swift phase, after which the nystagmus is called. From the graph it appears that the slow phase grows gradually swifter. The nystagmus needs some time to get started. Often there was a period of latency after the starting of the optokinetic stimulus. Gradually the head deviates from the central line towards the direction of the rotation of the field of vision. When the cylinder rotates more quickly the swift phase remains the same, but the slow phase has a much swifter course. The swifter the movement of the field of vision, the swifter the nystagmus, but the velocity can become so great that the pictures grow blurred, in which case optokinetic nystagmus cannot be engendered any more. When the cylinder stops the nystagmus continues for some time with smaller strokes, on this graph for seven to eight seconds. The post-rotatory nystagmus has the same direction as the optokinetic rotatory nystagmus. We see, therefore, that the optokinetic nystagmus is opposed to the direction of the rotation of the field of vision, the velocity of the rotation influences the slow phase.

Visser demonstrated that with the decerebrated pigeon an optokinetic nystagmus can also be generated, showing exactly the same characteristics as with the normal pigeon, but of much smaller intensity. In that case we have to deal with the subcortical form only. The considerable decrease of the optokinetic nystagmus as opposed to the vestibular one in the decerebrated pigeon does make it probable that for the normal pigeon the look-nystagmus is of some importance.

Fig. 13 is a typical example of the course of the optokinetic nystagmus of a decerebrated pigeon. Previous to the decerebration, which was performed nearly two months before this examination, the pigeon was also examined. At the time there was a very marked optokinetic nystagmus with wide strokes during the rotation. The post-rotatory reaction was very slight. After the decerebration it is striking (Fig. 13) that the period of latency at the start of the rotation is longer than with the normal pigeon. At first the strokes are hardly visible. Gradually they grow wider. They remain much smaller, however, than with the normal pigeon. It is striking that the post-rotatory nystagmus was still present, with this pigeon it lasts about five seconds in the light. This is probably explained by the fact that the decerebration eliminates the fixation. Owing to this the post-rotatory reaction can operate for a longer time.

In contrast with man the pigeon has a complete crossing of the optic nerve in the chiasm. When one eye of a pigeon is covered half

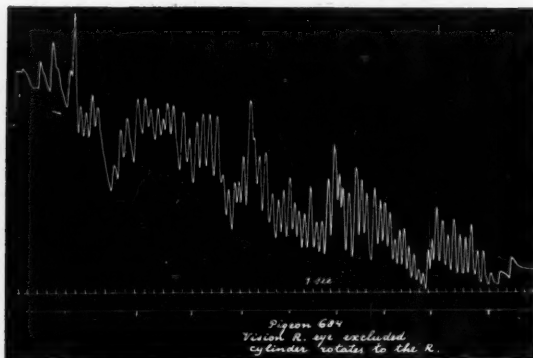


Fig. 14.—Optokinetic nystagmus. Normal pigeon. Right eye excluded. Cylinder rotating to the right.

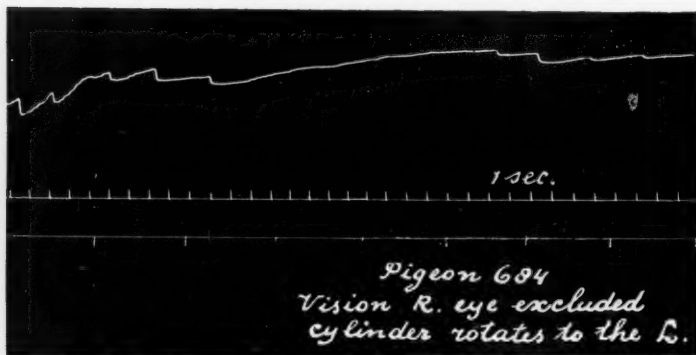


Fig. 15.—Same pigeon as Fig. 14. Optokinetic cylinder rotates to the left.

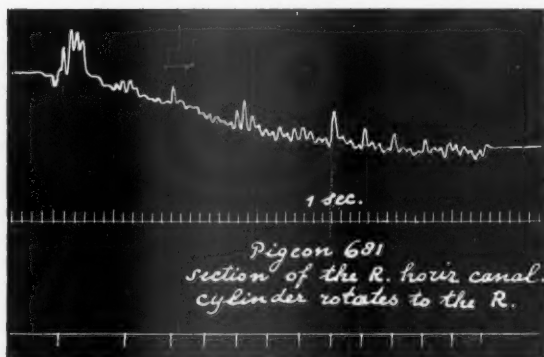


Fig. 16.—After section of the right horizontal canal. The optokinetic cylinder rotates to the right.

of the field of vision is eliminated and hemianopsia results. Records were made of 17 pigeons having one eye covered.

Figs. 14 and 15 show characteristic curves of a pigeon having its right eye eliminated. In Fig. 14 the field of vision moves to the right, and in Fig. 15 to the left. A great difference exists between these two curves. When the field of vision rotates to the right an obvious nystagmus is visible, so it is dependent from the uncovered left eye. On the other hand it appears that when the cylinder rotates in the opposite direction to the left, hardly any nystagmus exists. This is in agreement with the findings in certain cases of hemianopsia in man. On 17 pigeons the phenomenon was very clear 11 times. With the remaining ones the nystagmus was considerably decreased when the cylinder was rotated in the nasal direction.

It is remarkable that from two organs differing as much as the retina and the crista exactly the same reactions can be generated and that a more or less physiologic unity exists. When a pigeon with unimpaired vision is rotated around the vertical axis, e.g. to the right, the field of vision moves from the right to the left and so the right eye gives the strongest reaction. The same applies to the cristae. This is Ewald's second law. Under the same circumstances an ampullopetal endolymph-stream originates in the right horizontal canal, giving a stronger reaction than the ampullofugal endolymph-stream on the left side.

The correlation between the vestibular and the optokinetic nystagmus was made still clearer in the following way. The opto-

kinetic nystagmus of eight pigeons was recorded after section of one horizontal semicircular canal. A number of pigeons was examined after unilateral extirpation of the labyrinth. On the whole, these animals immediately after the operation have strongly decreased optokinetic reactions. The same applies to the vestibular reactions. Still the optokinetic reactions show an obvious difference between the right and the left. Fig. 16 shows the optokinetic nystagmus of a pigeon on which section of the right horizontal semicircular canal has been performed. The cylinder turns to the right, there is an obvious nystagmus with small strokes, there is also a head-deviation to the right. When the cylinder moves to the left no reaction at all takes place. Unilateral section of a semicircular canal, therefore, has the same effect as covering the eye on the same side. Previous examinations⁹ have made it clear that section of a semicircular canal results in elimination of the function of the crista concerned, owing to damage to these cristae. This probably causes changes in the vestibular centres which are also manifested in the course of the optokinetic nystagmus. In this way it was possible once more to demonstrate the close relation between the vestibular nystagmus and the subcortical form of the optokinetic nystagmus.

SUMMARY

In this investigation into the vestibular and optokinetic nystagmus of the pigeon a combination of the apparatus of Mowrer and Visser was used. In this way various combinations of vestibular and optokinetic reactions are possible. Mowrer's findings were completely confirmed. By the use of an optokinetic cylinder according to Visser the investigations could be enlarged upon. Mowrer could not yet take the fact into account that in the optokinetic nystagmus a cortical form and a subcortical one exist; both are probably of importance in the pigeon. During the rotation the vestibular and the optokinetic nystagmus reinforce each other. There is also an optokinetic post-rotatory reaction opposed to the vestibular post-rotatory reaction, so that after the rotations the reactions counteract each other.

There is a very great difference in the duration and the intensity of the vestibular reactions of various pigeons and of the same pigeon at different points of time. In this respect the cerebral cortex is of great importance. These great differences do not exist in decerebrated pigeons.

With the decerebrated pigeon the vestibular nystagmus is of much greater intensity and longer duration. The optokinetic nystagmus, however, has decreased. This is probably due to the elimination of the cortical optokinetic nystagmus.

The examination of the optokinetic nystagmus of normal and decerebrated pigeons affirmed Visser's investigations. We found that covering of one eye gives the same effect as section of one semicircular canal on the same side. This demonstrates once more the existence of a close relation between the vestibular nystagmus and the subcortical form of the optokinetic nystagmus.

It is pointed out, that various of these findings are important for the clinical examination of vestibular reactions in man.

NOORDERHAVEN 7.

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THE ALLERGIC FACTOR IN CHRONIC BRONCHO-PULMONARY INFECTIONS

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The behavior of the tissues of allergic individuals in response to infection or other form of stress is different from the response of the tissues of nonallergic people to similar insult.

As Kline¹⁷ has pointed out: Characteristics of infections in allergic people are rapid onset, violent course, and slow regression. Kline says the tissue changes in allergy may be considered under five headings: (1) functional, (2) inflammatory, (3) proliferative, (4) degenerative, and (5) necrotic. It is my belief that the necrotizing type of allergic reaction described by Kline is responsible for the development of many cases of bronchiectasis, bacterial proteins being the antigens responsible for the reaction. Upon reviewing the charts of 50 bronchiectatic patients,⁶ I found that 80% of them were allergic individuals. This corresponds closely with the figure given by Watson and Kibler,²⁴ who found that 82.6% of their bronchiectatic patients had allergic manifestations. The implication is not that sensitivity to foods or inhalants has much, if anything, to do with the development of bronchiectasis, but that persons with an allergic constitution are easily sensitized to bacterial proteins and thus apt to develop the necrotic type of allergic reaction described by Kline.

Kahn¹⁶ indicates that allergy is an expression of hyperimmunity and implies that it is an overaction of a physiologic mechanism. Kahn defines an allergic individual as one who develops immunity more readily, and to smaller quantities of an antigen, than does a normal individual. I agree with H. L. Williams²⁵ statement that, since the publication of "Tissue Immunity,"¹⁶ no evidence has been produced to indicate any inaccuracy in Kahn's conclusions.

One of the apparent benefits derived from the use of cortisone in these allergic individuals is its capacity to reduce the physiologic

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over-reaction to infection. More specifically, its use decreases the excessive mucosal edema and submucosal infiltration with leucocytes, thus widening the bronchial lumen, relieving the dyspnea, and permitting better drainage.

An instance of hyperimmunity or over-reaction to infection or other irritants may have no serious consequence if it takes place on a cutaneous surface, but when it takes place within the confines of a bronchial tube the resulting obstruction has serious consequences, causing dyspnea, bronchial obstruction, and often atelectasis, which, if long continued, can lead to bronchiectasis. This statement implies the need for early and adequate treatment by measures to be described, including bronchoscopic aspiration.

These remarks are made from the point of view of a bronchoscopist who perhaps has only a small knowledge of allergy, but who recognizes the importance of attempting to evaluate allergic factors in the diagnosis and treatment of chronic bronchopulmonary infections.

H. L. Williams²⁶ describes three related, but not identical types of allergy: (1) physical allergy; (2) bacterial or tissue allergy; and (3) humoral allergy. In my opinion, the chief difficulty of these individuals with chronic bronchopulmonary infection is the tuberculin type of bacterial allergy described by Swinford and Holman²¹ and by Scherago.¹⁸ In addition to the bronchopulmonary infection these patients frequently have foci of infection around the teeth, in the tonsils or sinuses. Adequate management of these patients with bacterial allergy includes elimination of all foci of infection. Effective treatment usually requires surgery plus bactericidal doses of penicillin. Grorud¹² says the antibiotic therapy of intrinsic asthma is not as valuable as was first anticipated. I think this is true because few men have used bactericidal doses. Bronfenbrenner² says the basic mechanism involved in bacterial allergy is identical with that of allergy to simple proteins. My remarks apply only to non-tuberculous pulmonary disease.

In an attempt to elucidate etiologic factors I studied the charts of 25 asthmatic patients for whom bronchoscopy was recommended either for diagnosis or for treatment. The age incidence is informative, one patient being in the first decade, one in the second, two in the third, one in the fourth, six in the fifth, ten in the sixth, four in the seventh. Thus 20 of the 25 patients were over 40 years of age. This is in line with the observations of Brown³ and Forman¹⁰ that allergic disease that begins after the age of 40 years is almost always due to bacteria. All of the 25 asthmatic patients for whom

bronchoscopy was deemed necessary had chronic suppurative bronchitis which indicates that the infectious factor must be vigorously treated if their symptoms are to be relieved. Four of the 25 in addition had bronchiectasis. In each of these four cases there was a clear history of pneumonia or pneumonitis which initiated the bronchiectatic symptoms. This is in line with my previous observation⁷ that patients having bronchitis or asthma or both never develop bronchiectasis unless they have had an episode of acute destructive pneumonitis. It also lends weight to the idea that bronchiectasis can be prevented if acute bronchopulmonary infections in allergic individuals are promptly and vigorously treated with bactericidal doses of the appropriate antibiotic or combinations of antibiotics. Large doses are necessary because many of the cocci have become relatively resistant to the action of penicillin.⁸ Thus the prevention of bronchiectasis is the serious responsibility of the physician who first sees an allergic individual with an acute bronchopulmonary infection. My usual formula for treatment of such patients is intramuscular injection of 800,000 units of fortified procaine penicillin every four hours, omitting the 4 A.M. dose, plus $\frac{1}{2}$ gram of streptomycin intramuscularly every 12 hours. In vitro studies have shown that mixtures of streptomycin and penicillin have a synergistic action. I have not placed much reliance on aureomycin and terramycin because they are bacteriostatic rather than bactericidal. This probably accounts for the relatively poor clinical results despite the high degree of in vitro sensitivity shown by most of the gram positive cocci to these two antibiotics. In addition, I think it is helpful to administer cortisone, 25 mgs. by mouth, every four hours to decrease the excessive allergic inflammatory reaction which causes bronchial obstruction and retention of exudate. The possible benefit deriving from combined use of cortisone and penicillin was suggested by Selye.¹⁰

I think it is bad practice to use antibiotics topically because of the danger of sensitization. I had several asthmatic patients who were made distinctly worse by penicillin aerosol inhalations so I quickly discontinued this type of therapy.

Five of the asthmatic patients had, in addition, Loeffler's syndrome or allergic pulmonary consolidation, of the type described by Hansen-Pruss and Goodman.¹⁴ Thirteen, or approximately half of these asthmatic patients with bronchitis, in addition had chronic suppurative sinusitis. The frequent coincidence of sinusitis and infectious asthma does not prove that one caused the other. My interpretation is that bacterial allergy is responsible for the chronicity of the infection in both the upper and lower respiratory tracts.

The indications for bronchoscopy in asthmatic patients can be considered under two headings: first, as a means of diagnosis; second, for therapeutic benefit by aspirating the viscid exudate which the patient has been unable to cough out. In regard to diagnosis, bronchoscopy is of value in ruling out other causes of wheezing. It also gives a rather clear idea of the degree of bronchitis present and the degree of mucosal thickening, but more important, it furnishes an uncontaminated specimen of endobronchial exudate for smears and cultures, including sensitivity cultures, which I think are important in indicating the choice of antibiotic to be used, and the dose which will probably be necessary to eradicate the infection. In this series of 25 cases, 16, that is 64%, had at least temporary therapeutic benefit by removal of the gummy endobronchial exudate. This benefit was especially noticeable in the patients who had Loeffler's syndrome.

Three of the patients were made distinctly worse by bronchoscopic examination because the procedure induced severe acute bronchospasm, and one of these patients died shortly after the bronchoscopic examination. This bronchoscopy was not performed by me. On reviewing the chart it seems evident that bronchoscopy should have been deferred until his extreme state of exhaustion and dehydration had been corrected by medical means.

Three of the patients with Loeffler's syndrome were thought from the radiologic examination to have tuberculosis, but the repeatedly negative sputum examinations and prompt clearing of the pulmonary densities ruled out the latter.

A review of the case histories of several of these asthmatic patients referred for bronchoscopic examination will, I think, illustrate the infectious bronchitis, which in my opinion is the chief etiologic factor in those patients who require bronchoscopic examination.

REPORT OF A CASE

CASE 1.—I. Y., aged 52, was admitted on our service in February, 1950. She said that her asthma began after she had an attack of "influenza" 20 years ago. Her asthma had been intermittent and there might be an entire year when she did not have any wheezing or other evidence of asthma. Three months prior to admission she developed bronchitis and marked persistent wheezing with difficulty in expiration. She had a loud expiratory wheeze which suggested some type of bronchial obstruction other than bronchial asthma. There were musical rales throughout both sides of the chest. Bronchoscopic examination showed rather marked diffuse congestion and

edema of the tracheobronchial mucosa and the mucosa was considerably thickened as evidenced by the fact that the spurs were much wider than normal. A moderate amount of mucopurulent exudate was aspirated and sent to the laboratory for smears and cultures. There was no visible obstruction of any of the branch bronchi other than the partial obstruction produced by generalized mucosal edema and submucosal infiltration. Smears of the endobronchial exudate showed many pus cells, but only a few eosinophils. Culture showed a hemolytic staph aureus which grew on a 3 unit penicillin plate, was inhibited on a 5 unit penicillin plate, and on the 5 microgram streptomycin plate. She was treated with fortified procaine penicillin, 400,000 units every three hours and streptomycin, 0.7 grams every 12 hours for a period of seven days. She was totally free from cough and wheezing when she left the hospital at the end of a week's treatment, so it seems evident that the appropriate antibacterial therapy was effective. Bronchoscopy was useful in this case in focusing attention on the bacterial bronchitis and in furnishing an uncontaminated specimen for smears and sensitivity cultures. Seventy skin tests were made by the method described by Walker,²³ and all were negative except positive reactions to timothy and several of the June grasses. She had no increase in symptoms during the summer months, so it is evident that she did not have clinical sensitivity to pollens. X-ray examination of her chest showed no abnormality other than the presence of a few small calcium deposits in the region of the right middle lobe. She had no recurrence until she developed bronchitis seven months later.

CASE 2.—E. S., aged 44 years. This patient had an acute exacerbation of chronic suppurative bronchitis. He had bronchial asthma of approximately ten years' duration and his asthma had been worse for two weeks prior to admission to the hospital. X-ray examination showed marked bullous emphysema. Bronchoscopic examination showed acute diffuse congestion of the tracheobronchial mucosa and several cc of thick mucopus were aspirated. Introduction of the bronchoscope caused considerable increase in his bronchospasm and produced cyanosis. Bronchospasm developed even though he had been given aminophyllin intravenously and adrenalin subcutaneously just prior to the bronchoscopy. The events in this case report suggest that bronchoscopic aspiration should be deferred as advised by Hansel¹³ until the acute bronchitis is controlled by medical means. This patient was treated with fortified procaine penicillin, 400,000 units every six hours, and streptomycin, 0.7 grams every 12 hours, as well as by aminophyllin administered by slow intravenous drip twice daily. His temperature, which had been 101,° came down

to normal and he left the hospital much improved at the end of five days.

CASE 3.—H. C., aged 67. This patient had recurrent asthma since she was 38 years of age, but had had asthma almost continuously for six months prior to admission, required adrenalin injections for relief and had been coughing up one ounce of yellow sputum daily. There were loud wheezes throughout both sides of the chest. X-ray examination showed considerable emphysema and segmental atelectasis in both lower lobes. Bronchoscopic aspiration yielded several cc of extremely thick, gummy, yellow mucopus. There was moderate diffuse congestion and edema of the tracheobronchial mucosa. Smears from the endobronchial exudate showed many gram positive cocci and many pus cells. No eosinophils were seen. Culture showed a strep viridans which grew freely on the 1 unit penicillin plate, but which was inhibited by the 3 unit penicillin plate, as well as by the 5 micrograms streptomycin plate. She was treated with fortified procaine penicillin, 400,000 units every six hours, and streptomycin, 0.75 grams every 12 hours for a period of seven days. Her asthmatic symptoms cleared up completely and the patient said her health was the best it had been in the preceding two years. A follow-up report 15 months later said that she had had no recurrence of asthma, had not needed a doctor and enjoyed doing all of her own housework.

CASE 4.—K. Q. This patient, aged 52, had asthma, which developed immediately following an acute upper respiratory infection two months prior to admission. The asthma has been so bad that she had to sit up in a chair all night. She had a moderate amount of mucopurulent sputum. She never had any previous asthma or any type of allergic symptoms. She was treated in another hospital with penicillin, epinephrine, and oxygen, without relief, so her doctor thought her asthma might be on a cardiac basis and gave her digitalis. There was no family or personal history of allergy. The blood count showed 24% eosinophils. X-ray examination on March 11, 1951, revealed consolidation in the superior segment of the left lower lobe. Bronchoscopic examination on March 15 showed congestion and thickening of the bronchial mucosa with the bronchial lumen markedly diminished on expiration. About 2 cc of tenacious yellow mucopus were aspirated. The bronchologist made a diagnosis of Loeffler's syndrome and advised treatment with ACTH. The second x-ray examination on March 17 showed 40% clearing in the shadows of infiltration and consolidation in the left lung and a new area of increased density in the right middle lobe. Third x-ray examination on March 24 showed normal looking lungs. She had fever up to

101° prior to bronchoscopic aspiration and administration of ACTH. Thereafter her temperature remained strictly normal. In addition she received aminophyllin by slow intravenous drip. No aminophyllin was needed after the 17th, that was two days after bronchoscopic aspiration and beginning of administration of ACTH. A follow-up report three months later stated that she had not had any recurrence.

A review of the charts of these patients with chronic suppurative bronchitis and asthma is depressing because so many of them have recurrences. Nearly all of them had very gratifying response to treatment with large doses of penicillin and the other measures described over a period of seven to ten days. Most of them would remain free from symptoms for periods varying from one to six months, until another respiratory infection again precipitated asthma. In other words, treatment with penicillin does nothing to prevent the next infection. The same temporary type of benefit is evident in the patients who receive either ACTH or cortisone.

On the title page of the book by Sir Almroth Wright,²⁷ entitled, "Studies on Immunisation, Second Series," is the statement: "The physician of the future will be an immunisator." I must confess that vaccine therapy is one type of treatment I have neglected in the management of cases of this type since penicillin became available. My recent reading has given me the conviction that it is a type of treatment that can benefit some of these patients. The effectiveness of vaccine therapy has been pointed out by Cooke,⁵ Clerf,⁴ Wright,²⁷ Brown,³ Silcox,²⁰ Gay,¹¹ Unger,²² Zinsser,²⁸ Eagle,⁹ Benson,¹ Heise¹⁵ and many others. I think vaccine therapy should be used when removal of foci of infection and high dose antibiotic therapy have failed to cure the patient.

CONCLUSIONS

Asthma that begins after the age of 40 is usually due to infectious bronchitis and is due to the tuberculin type of bacterial allergy. Adequate management of these patients having bacterial allergy includes elimination of all foci of infection.

Bactericidal doses of penicillin combined with the usual antiasthmatic measures will frequently clear up the pulmonary infection if other foci of infection are eliminated by surgery.

Bronchoscopic examination is of value for both diagnosis and treatment. It focuses attention on the infectious bronchitis which is apt to be minimized or overlooked in asthmatic patients.

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LXXXIII

FISH BONES IN THE ESOPHAGUS

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Endoscopy has gained an important and secure place in the diagnosis and treatment of diseases of the bronchopulmonary tree and the esophagus. However, the foreign body, which originally served to inspire the development of bronchoscopy and esophagoscopy, has been relegated to a place of lesser interest. Yet, the recognition of the presence of foreign bodies, particularly the non-opaque variety, their localization and removal, and the management of associated complications pose some of the most difficult problems and often present some of the most gratifying results.

Opaque foreign bodies in the cervical esophagus, as a rule, are readily diagnosed with the aid of x-ray studies. Non-opaque foreign bodies are more difficult to discover and may require indirect methods of demonstration. There are instances in which the diagnosis of a non-opaque foreign body is made on symptomatology alone, and the decision for performing an esophagoscopy must be reached by clinical judgment. An appraisal of x-ray findings in a group of cases in which the roentgenographic diagnosis of foreign bodies is equivocal is very desirable in order to determine the value of diagnostic methods available.

The teaching has been for some time that fish bones in the esophagus are usually transparent to x-rays. Hence, roentgen ray examination of the esophagus for fish bones has been considered of little value. When roentgen examination of the esophagus (invariably the cervical area) for a fish bone failed to show an opaque body and when symptoms persisted, it has been necessary to resort to esophagoscopy as a diagnostic procedure. This, of course, is far from an ideal situation and is not in full accord with my own clinical experience.

With the foregoing in mind, this study was undertaken to determine whether fish bones in the esophagus are or are not as frequently translucent roentgenologically as it is commonly believed. Accord-

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ingly, this presentation is based on an analysis of a series of fish bone foreign bodies of the esophagus in a general hospital and a roentgenological study made on fish rib bones in a cervical phantom which closely simulates the neck and superior mediastinum in tissue density to x-rays.

The roentgenological studies were carried out in association with Dr. Arnold L. Bachman.

LITERATURE

It is rather striking that in articles on foreign bodies in the esophagus little attention is directed to the radiopacity of fish bones. X-ray studies, as a rule, do not enter into the consideration of the diagnosis of fish bones in the esophagus. When on rare occasions this problem is discussed, it is the inadequacy of roentgen diagnosis that usually is stressed. It is likely that unsatisfactory experiences with x-ray techniques in the early part of this century are responsible for this attitude.

Wilson¹ in 1925 commented: "The fish bone, and often the dangerous sickle shaped haddock bone, is one of the commonest of the various foreign bodies which become lodged or impacted in the human gullet. In such cases the information obtained from an x-ray screen examination or from radiograms is generally unsatisfactory and may be misleading." He further emphasized that the x-ray findings are frequently negative when fish bones are present in the esophagus. Somewhat later (1933), Carlsund² was of the same opinion when he said that the x-ray is not very helpful in detecting fish bones in the esophagus and in support of his view quoted Teschen-dorff (1928) who also believed that the roentgen ray was of little value in the diagnosis of fish bones in the esophagus.

Jackson and Jackson,³ in their book "Foreign Body in Air and Food Passages," (published in 1934) made the following two statements supporting the generally expressed view: "The bones of fish do not ordinarily cast any shadow even at first lodgment when there are no obscuring pathologic shadows." And again, "Fish bones as a rule are not dense enough or not sufficiently thick to cast a shadow."

Skarby⁴ (1944) in the Scandinavian literature, on the other hand, regarded x-ray examination of distinct value in the diagnosis of fish bones in the esophagus. He quoted Minnegerode, Stegemann, Spiess, Biering, Kindler and others as investigators who agreed with this point of view. However, he also quoted a number of authors whose opinion it was that x-ray was not practical for the diagnosis

of fish bones. These were von Hacker, Teschendorff, Köhler, Ulrich and Killian.

Skarby also conducted x-ray studies on a number of local Scandinavian and European fish to determine the roentgen density of their bones. Placing the fish in water in an aluminum container and using the special Laurell¹² technique for visualization, he found that the bones of all the fish were visible, even the finest.

In view of the poor regard in which direct roentgen visualization of fish bones in the esophagus was generally held, indirect methods for the detection of the fish bone have been favored. Swelling of the prevertebral tissue or the presence of gas in these tissues has been considered suggestive of lodgment of a foreign body. Brown⁵ (1945) has suggested that one should be suspicious of a foreign body whenever the diameter of the esophageal shadow exceeds that of the vertebra or trachea. The coating of a suspected foreign body in the esophagus by an opaque substance such as barium, bismuth and lipiodol has been a widely used indirect method for the demonstration of fish bones. Manges⁶ (1927) advised the use of bismuth suspensions. Barium emulsion has been regarded as a valuable contrast medium by Kjellberg⁷ (1935), Scott and Moore⁸ (1936), Brown⁵ (1945) and Johnstone⁹ (1947). Carlsund² (1933) advocated the use of barium emulsion followed by swallowing a small amount of water. The latter was to wash away all the residual barium from the normal esophageal mucous membrane, leaving small amounts of barium clinging to the foreign body to outline it. Wilson¹ (1925) advised swallowing wool soaked in barium but this technique has been abandoned by most workers in this field. Utrata¹⁰ (1947), on the other hand, opposed the use of barium in the belief that it may obscure the foreign body during esophagoscopy and thereby militate against successful removal. However, this objection can be obviated by drinking a considerable amount of water which will wash away all the residual barium. Reincke¹¹ (1928) has found lipiodol useful to coat foreign bodies such as fish bones.

MATERIAL

This study of a series of patients who had swallowed fish bones was made for the purpose of determining the visibility of these bodies in the esophagus on x-ray examination. Records were obtained of 185 cases of foreign bodies in the esophagus collected over a period of 15 years (1933-1948) from the ward and private services of The Mount Sinai Hospital, New York. These cases may be grouped as follows (Table I):

TABLE I.

CHARACTER OF FOREIGN BODY	NO. OF CASES
Fish bones	56
Chicken bones	55
Meat bones	15
Metal (coins, dentures)	47
Others	12
Total	185

Of the 185 patients, 56 were admitted to the hospital suspected of harboring a fish bone foreign body in the esophagus. Among them, 45 were females and 11 males, all showing a fairly uniform age distribution (Table II).

TABLE II.

AGE	MALE	FEMALE
0-10	0	0
11-20	1	2
21-30	0	1
31-40	3	11
41-50	1	15
51-60	5	5
61+	1	11
Total	11	45

The outstanding symptom in 36 patients was persistent pain in the neck, located laterally or in the area of the suprasternal notch. In one patient, the pain radiated constantly to the ear, and in three patients the pain appeared on movement of the neck, particularly into extension. A sticking sensation as distinguished from pain was present as a complaint in ten patients. Thirty patients suffered from varying degrees of dysphagia. In several instances there was complete inability to swallow. Tenderness over the neck, particularly

behind the thyroid cartilages and over the suprasternal notch, was noted in 22 instances. Marked pooling of saliva in the pyriform fossae was observed in a small number of patients.

In 40 instances of the 56 cases, a fish bone was demonstrated at esophagoscopy or operation. The fish bone was found at the cricopharyngeus or within 2 cm below the cricopharyngeus in 17 cases, and in 22 other patients the foreign body was located in the upper esophagus above the thoracic inlet. Of these 40 cases, roentgen or direct evidence of a fish bone was found in 30 patients (75%).

The 56 cases were also analyzed with the aim to determine the significance of indirect roentgen findings, such as prevertebral widening and the presence of gas in the prevertebral tissues. Prevertebral widening due to edema was found associated with a visualized fish bone in 12 instances, gas with a visualized fish bone in five instances. Prevertebral widening when no fish bone was visualized was found in five patients, gas in one patient (Table III).

TABLE III.

X-RAY FINDINGS	NO. OF PATIENTS
Direct visualization of fish bone	30
Indirect findings (prevertebral widening, gas)	
Fish bone visualized	
1. None	18
2. Prevertebral widening	12
3. Gas	5
Fish bone not visualized	
1. None (x-ray negative)	5
2. Prevertebral widening	4
3. Gas	1

There were 16 patients whose symptoms, following the ingestion of fish, warranted esophagoscopy or operation, but from whom no fish bone was recovered. These showed the following x-ray findings (Table IV):

TABLE IV.

X-RAY FINDINGS	NO. OF PATIENTS
Direct visualization of fish bone	2
Indirect findings (prevertebral widening, gas)	
Fish bone visualized	
1. None	2
Fish bone not visualized	
1. None (x-ray negative)	9
2. Prevertebral widening	5
3. Gas	3

Thus, in 21 cases, x-ray studies showed varying degrees of prevertebral widening. In considering the relation of prevertebral widening to the duration of sojourn of the foreign body, it was found that widening appeared in seven cases in 24 hours after swallowing the fish bone and in one case the edema developed within three hours (Table V).

TABLE V.
PREVERTEBRAL WIDENING

DURATION OF SOJOURN	NO. OF PATIENTS
Up to 12 hours	3
12 to 24 hours	4
24 to 48 hours	2
Over 2 days	9
Unknown	3

When correlating prevertebral widening with clinical complications, such as periesophageal abscess or mediastinitis, it was noted that gas was usually encountered when a complication existed. Thus, of 12 cases with widening alone, a complication was found in only one case, while all nine cases had a complication when gas was present in addition to widening (Table VI).

TABLE VI.
PREVERTEBRAL WIDENING ASSOCIATED WITH CLINICAL
COMPLICATION.

	CASES	WIDENING ALONE	WIDENING AND GAS
Foreign Body with Complication	10	1	9
Foreign Body without Complication	11	11	0

Of the 21 patients with prevertebral widening, a fish bone was seen on esophagoscopy in 16 instances and visualized on roentgenograms in 12 instances.

Of the 56 patients with symptoms after ingestion of fish, ten (18%) had serious complications resulting from injury caused by fish bones. Of the ten complications, there were four cases of periesophageal abscess, three cases of mediastinitis, three cases of perforation of the esophagus. Two of these patients died; each had suffered from diabetes.

Roentgen examination for chicken bones in the esophagus has been regarded as a valuable procedure for the diagnosis of these foreign bodies as they usually have been considered opaque. Therefore, a comparative analysis of 55 cases with symptoms following the ingestion of chicken bones is of interest.

There were 47 patients in whom a chicken bone was demonstrated endoscopically or at operation and who had an x-ray examination. Visualization of the chicken bone on roentgenography was possible in 34 patients (72%). The visualization in this series was lower than in the series of fish bone cases (75%). There were ten cases of perforation caused by chicken bones and resulting in either a periesophageal or mediastinal complication. They constitute 18%, a percentage identical with that of complications caused by fish bones.

EXPERIMENTS

Rib bones of varying thickness and size were removed from different types of fish and employed in this study. The bones were obtained from uncooked and cooked fish. The thickness and shape of the specimens were noted and the bones were radiographed by themselves and in phantoms to simulate clinical conditions. Two types of phantoms were used: (1) A slab of meat about 7.5 cm in thickness in the center of which were placed the bones to be x-rayed,

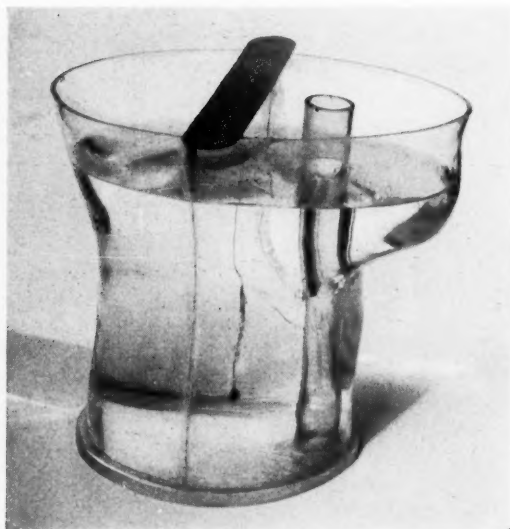


Fig. 1.—Cervical phantom filled with water. Fish bones for radiographic study were attached to radiolucent cotton thread with scotch tape. Small lead weight at bottom of thread.

(2) a hollow plastic model simulating the lower face, neck and thoracic inlet (Fig. 1). The neck portion of the phantom measured 10 cm in diameter. A small cylinder 1.5 cm in diameter was placed in the anterior part of the hollow shell to simulate the laryngotracheal airway. This hollow neck-shaped plastic tank was then filled with water except for the cylindrical pseudo-laryngotrachea. The phantom when filled with water closely simulates the density of neck tissues and therefore was ideally suited for noting the radiability of the various bones. The shape of the cervical phantom and the use of water as the medium created the same factors for radiation scattering and absorption as are encountered clinically.

Most of the experiments were conducted with the cervical phantom. The bones to be radiographed were attached to a completely radiolucent cotton thread and suspended either in the water or in the pseudo-laryngotracheal plastic air column standing in the water. Usually radiographs of the bones were made both in water and in the air column.

Different radiographic techniques were tried, using both the meat and cervical phantoms. The effect of under-exposure, optimum-

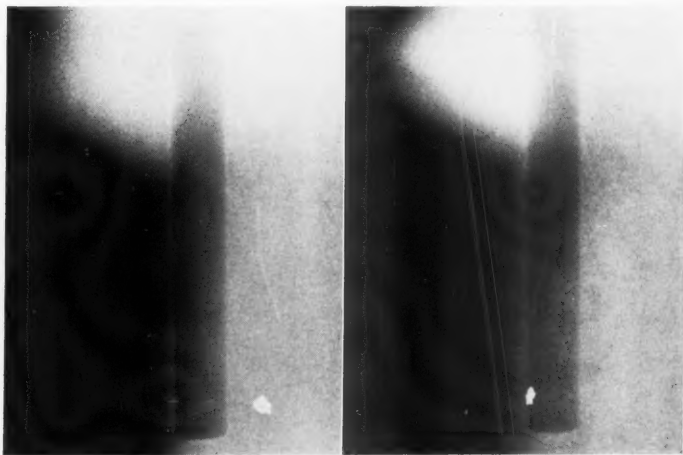


Fig. 2.—Radiograph of cervical phantom with pompano bone above and cod bone below suspended in water. Pompano bone is invisible and cod bone clearly visible.

Fig. 3.—Same as Fig. 2 with bones suspended in air column. Both bones are better visualized in this medium.

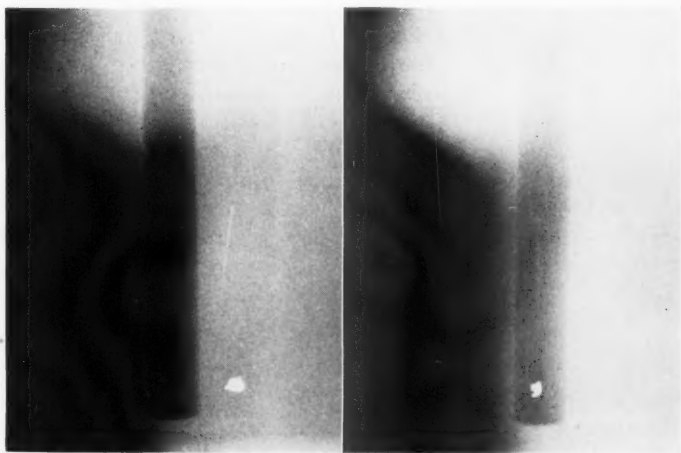


Fig. 4.—Radiograph of cervical phantom with mackerel bone above and porgie bone below suspended in water. Mackerel bone is invisible and porgie bone clearly visible.

Fig. 5.—Same as Fig. 4 with bones suspended in air column. Both bones are better visualized in this medium.

exposure and over-exposure techniques on the radiographic demonstration of the same bones was studied. Trials of Laurell's¹² high-voltage, long object-film distance technique for faintly visualizing bones were also made, employing the cervical phantom.

Various bones from 21 different types of fish were studied. With one exception (smelt), more than one bone from more than one sample of each kind of fish was radiographed (Figs. 2 to 7). All varieties excepting the butterfish, pompano and trout were definitely visualized.

Cooking of bones prior to x-ray was done in about half the cases. There was no difference noted in the radiographic visibility of these bones as compared with the uncooked bones.

Technique played a significant role in the above experimental studies. The best results were obtained by employing a very short exposure (1/20-1/10 sec.), with small coning, small focal spot and an x-ray negative slightly on the dark side (Fig. 8). The latter was distinctly preferable to a light film. In several instances (bluefish, fresh salmon, smoked salmon) a bone which was not visible on a somewhat light or very dark film became clearly visible on a slightly dark negative. Laurell's special long object-film distance technique was not found consistently better than a good standard moderately dark technique and does not appear warranted for general use. In one instance (halibut), a rather dense flat bone was x-rayed "en face" and its density in the film was much less than seen with three other halibut bones that were radiographed. However, most flat foreign bodies in the upper esophagus lie in the coronal plane. Therefore, they are seen "on edge" in the lateral radiograph of the neck and thus appear much denser than when visualized "en face." Occasionally fish bones may not lie in a true coronal plane but are placed in an oblique one. Accordingly, it may be worthwhile to take x-rays in slightly oblique positions when fish bones are suspected and they fail to be seen in roentgenograms taken in standard lateral positions.

This point of view is well illustrated by investigations conducted on a haddock bone in the cervical phantom after it was removed from an esophagus. Roentgenograms of this bone in the esophagus showed a linear foreign body (Fig. 9). On removal it was found to be a large fan-shaped gill bone (Fig. 10). Roentgenographs of this bone in the cervical phantom clearly show the variations in densities that can be obtained in the different positions (Figs. 11 and 12).

Knowledge of the expected densities of fish bones on roentgenograms can be helpful in evaluation of the clinical picture of a patient

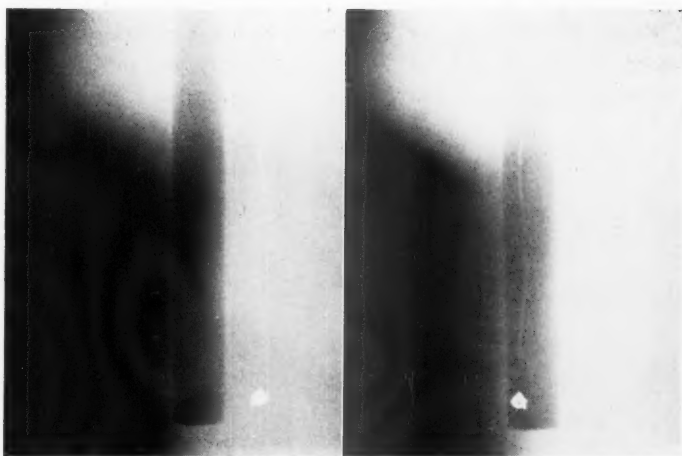


Fig. 6.—Radiograph of cervical phantom with haddock bone above, bass bone in the middle and fluke bone below suspended in water. All three bones are clearly visible.

Fig. 7.—Same as Fig. 6 with bones suspended in air column. All bones are better visualized in this medium.

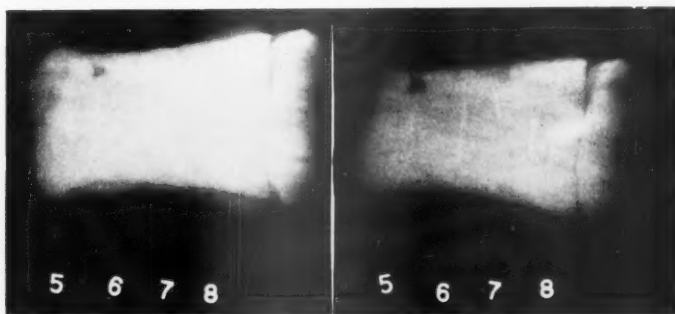


Fig. 8.—Radiograph of meat phantom with fish bones placed between layers. No. 5—codfish, no. 6—bluefish, no. 7—mackerel, no. 8—smoked salmon. Note that slight over-exposure of radiograph on right produced better visualization of the bones than slight under-exposure of radiograph on left.

in whom lodgment of a fish bone in the esophagus is suspected. The expected densities of studies of the rib bones are listed in Table VII. It is believed that the bones in groups I and II can be diagnosed by x-ray visualizations while the bones in group III may not be suitable for roentgenographic diagnosis.

TABLE VII.
EXPECTED X-RAY DENSITY OF BONES

I. Radiopaque	II. Moderately radiopaque
Bass	Fresh Salmon
Codfish	Smoked Salmon
Flounder	Yellow Pike
Fluke	
Gray Sole	
Haddock	
Halibut	III. Little or no radiopacity
Porgie	Bluefish
Red Snapper	Butterfish
Sea Bass	Mackerel
Smelt	Pompano
Striped Bass	Trout
White Perch	

COMMENT

A review of complaints of patients harboring fish bones in the esophagus discloses that persistent localized pain in the neck, particularly on swallowing, is still a reliable symptom indicative of either the presence of a fish bone or local damage caused by the bone. This is especially true when pain is associated with a sticking sensation. On occasions, if the fish bone cannot be demonstrated by direct x-ray visualization, it may be necessary to perform an esophagoscopy as a further diagnostic procedure.

The significant fact demonstrated by this study is that direct roentgen visualization of the fish bone was possible in 30 of 40 patients (75%) in whom a fish bone was found at esophagoscopy or operation. In 39 of the 40 proved cases, the bone was located above



Fig. 9.—Lateral radiograph of neck showing haddock bone in cervical esophagus.

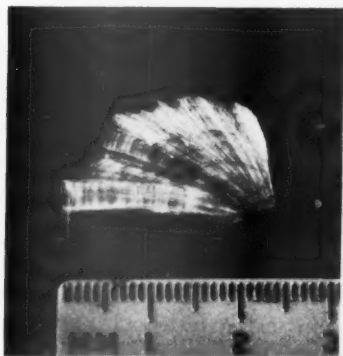


Fig. 10.—Photograph of haddock bone (Fig. 9) after removal.

the thoracic inlet or sternal notch. Thus, because fish bones are commonly lodged above the thoracic inlet, they should be visible on x-ray if they are radiopaque. On the other hand, even radiopaque bones can be obscured by adjacent structures if located below the thoracic inlet.

By way of comparison, it is interesting to note the findings of 55 cases in which symptoms followed the swallowing of chicken or chicken bones. It is generally regarded that chicken bones are readily visible on roentgenography. An opinion is universally expressed that roentgen examination for chicken bone is much more reliable than for fish bone. In this series there was little difference in radiopacity of chicken bones (72%) and fish bones (75%).

Indirect visualization of fish bone foreign bodies is stressed in the literature. The indirect findings which have been emphasized are widening or edema of the prevertebral tissue, the presence of gas in these tissues, demonstration of the foreign body by contrast material and the displacement of the esophageal lumen revealed also by contrast material. By widening is meant swelling or enlargement of the prevertebral tissues greater than the adjoining cervical vertebra. The information that can be gathered from these indirect methods is obviously quite substantial, and in this series contrast material might have been used to good advantage more regularly in those cases in which the fish bone was not visualized directly.

In this study prevertebral widening or edema was observed on roentgenography in 21 cases. Prevertebral widening appeared within 24 hours after ingestion of the fish bone in seven cases, and edema was observed in one case as early as three hours after swallowing the foreign body. Thus, it is obvious that edema can develop quickly and early in the sojourn of the fish bone.

It was also observed that widening alone is not indicative of the gravity that has sometimes been commonly associated with this condition. Of the 12 cases which showed widening alone, only one was associated with a complication. This patient had a periesophageal abscess in connection with a fish bone perforation. Endoscopic removal of the fish bone followed by antibiotic therapy resulted in recovery. On the other hand, all 9 patients who had gas in the prevertebral tissues in addition to widening suffered from a complication. This reaffirms the view which has long been accepted, that gas in the prevertebral tissues unquestionably indicates a complication.

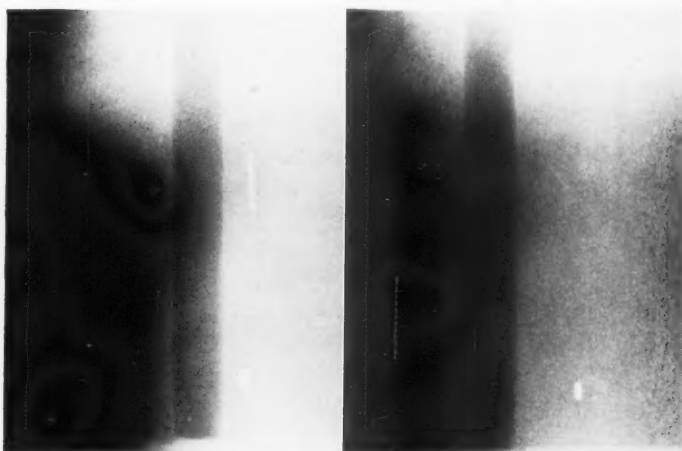


Fig. 11.—Same haddock bone (Fig. 10) radiographed on edge in cervical phantom. Note similarity to clinical x-ray (Fig. 9).

Fig. 12.—Same haddock bone (Fig. 10) radiographed "en face" in cervical phantom. Note diminished visibility as compared with Figs. 9 and 11.

Most distressing is the high incidence of serious complications noted in this series resulting from the trauma induced by the fish bones. Ten or 18% of the patients, equalling the frequency of complications among the chicken bone cases, developed complications. Five of these patients were subjected to external operative procedures. In one patient, a large periesophageal abscess, demonstrated by a fluid level in the roentgenogram, was drained and cured by endoscopic intervention. It may be significant that all the complications occurred before 1946, or before the era of intensive antibiotic therapy.

The study of the radiopacity of bones of fish found in the north-eastern states was made possible by the availability of a cervical phantom, used for depth dose determination in x-ray therapy. The cervical phantom, when filled with water, produced a density to x-rays comparable to the tissues of the neck and was, therefore, especially adaptable for this type of investigation.

It is believed that information concerning the radiopacity of bones of fish from this part of the country or similar data from different sections of the country, should be of practical value in the recognition of fish bone foreign bodies in the esophagus. In obtain-

ing the history from a patient who has swallowed a fish bone, the kind of fish ingested should be noted. Knowledge of the kind of fish ingested can often be helpful in the diagnosis and the management of such patients.

SUMMARY AND CONCLUSIONS

In this series of 185 cases of esophageal foreign bodies, 56 patients suffered from symptoms following the ingestion of fish. In 40 of these patients, a fish bone was demonstrated at esophagoscopy or operation. Diagnosis by roentgen examination occurred in 30 patients or in 75% of the proved cases.

Of 47 cases of chicken bone foreign bodies, the presence of which was established at esophagoscopy or operation, a bone was demonstrated by direct x-ray visualization in 34 cases (72%).

The bones from 21 different fish indigenous in the northeastern part of this country were studied roentgenographically. This was accomplished by suspending the bones in a cervical phantom filled with water. Sixty individual examinations were made. It was found that uncooked and cooked bones yielded the same visibility. X-ray technique played a significant role in the demonstration of these bones. Fish bones whose radiopacity can be expected to be of diagnostic value are bass, codfish, flounder, fluke, gray sole, haddock, halibut, porgie, red snapper, sea bass, smelt, striped bass, white perch. Fish bones which can be expected to be radiopaque but to a lesser degree are fresh salmon, smoked salmon, yellow pike. Fish bones which may produce faint to no opacity are bluefish, butterfish, macerel, pompano, trout.

This study provides information which strongly suggests that the more common impression that roentgen examination of fish bones in the esophagus is unreliable because they are infrequently radiopaque is not justified. The data gathered from an analysis of instances of fish bone foreign bodies in the esophagus in a general hospital and from experimental roentgenological studies on rib bones from known fish rather indicates that, with roentgen techniques used at the present time, there is a high percentage of positive radiopacity of fish bones. Thus, greater reliance on roentgen diagnosis in cases of suspected fish bones in the cervical esophagus is warranted.

Other information presented in this investigation concerns symptomatology, indirect evidence of fish bone lodgment and trauma, complications associated with fish bone ingestion and x-ray factors necessary to obtain optimum visualization of the fish bone.

1050 PARK AVENUE.

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LXXXIV

ON THE FORMATION OF THE OTOLITHS

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Numerous investigators have tackled the problems turning up in the study of the formation, deposition and function of the otoliths. Yet the more important problems are still obscure. The presence of the otoliths has been known for a very long time. Scarpa was the first to correlate them with the function of the labyrinth and designate them as "hearing stones." Merely a brief account will be given here of the more important investigations reported on the origin and deposition of the otoliths (e.g. Belonoschkin, Wittmaack, Studnicka, Donadei, v.d. Stricht, Herzog and Nishio). On the basis of these works it is the prevailing opinion that the otoliths are intimately related also histogenetically to the surrounding gelatinofibrillary network. The macular cells, or the capillaries below the macula, are believed to "secrete" calcium salt into the aqueous endolymph at a very early state of development. The secreted amounts of salts are supposed to be present in a soluble form and some obscure causes (shift in pH? according to Wittmaack) are then assumed to bring about their precipitation in the form of small, though visible crystalline granules. The granules increase in size and merge together, or they may form individual centers of growth (Nishio). The resulting otolithic mass may either form one solid mass or it may consist of a heap of small granules (e.g. Elasmobranchii and mammals).

In the fresh, transparent eggs of *Betta splendens* (a carp) Nishio has observed the formation of calcium granules as well as their growth and agglutination in the labyrinth. He, and later Herzog, wondered at the considerable amount of calcium salt present at this early juncture in the labyrinth in an animal which did not yet contain any amount of calcium salt worthy of mention in the rest of the organism. So presumably by far the greater part of the total calcium content of the animal at this early juncture was deposited in the labyrinthine membranous sacs (Herzog).

Concerning the Elasmobranchii, Nishio demonstrated that many of the otoliths (otoconia) in the sacculus deviated from the other granules and showed a striking resemblance to the particles of Medi-

terranean bottom sand of lava character. Similar observations were reported by Werner. Neither investigator, however, employed any physical method of analysis to prove their statement indisputably.

The present material consists of eight fetal sharks (*acanthias vulg.*) removed from the maternal uterus *in vivo* and six adult sharks. The material was fixed *in vivo* in formalin and mercuric chloride solution, embedded in paraffin, cut in sections of seven to 12 μ and stained with hematoxylin dyes. The endolymphatic duct was studied partly in serial sections from the entire labyrinth, partly in sections from endolymphatic ducts that were dissected out. The labyrinths of the adult sharks were fixed *in vivo* in the same fluid. Other technical data are mentioned below.

The otoliths in the shark labyrinth seem to be of different origin. We meet with endogenous otoliths partly produced *in loco*, i.e. above the maculae in early stages of development, with endogenous otoliths produced elsewhere in the labyrinth (postnatally) and exogenous otoliths introduced into the labyrinth from the outside and situated above the maculae after birth (grains of sand).

ENDOGENOUS OTOLITHS

In fetuses of more than five cm in length from snout to tip of tail the labyrinth is well developed even though the lining epithelium is not fully differentiated in its final form. In sections of such fetuses no definite evidence of the presence of gelatinous macularia is found (disappeared during the preparation of the labyrinth?) but numerous small, apparently crystalline granules are seen immediately above the site of the maculae. When a drop of hydrochloric acid is placed on these granules gas is produced, indicating that the granules may consist of calcium carbonate. These granules are not found elsewhere in the endolymphatic space. They grow in size during the intrauterine growth of the fetus and when gelatinous substances later appear over the maculae in the sections, the granules are found within the meshes of these gelatinous substances. The granules are birefringent. No doubt they correspond closely to the granular calcium deposits above the maculae described by many authors and believed to have originated *in loco*. Details concerning the nature of their formation are still lacking.

In the wall of the endolymphatic duct the writer found numerous "vacuoles" which, by the micro-incineration, crystal-optic and roentgenographic examination were proved to contain inorganic crystalline salts in non-calcified ground substance. These vacuoles are seen deeply in the epithelium lining the duct, in the middle of

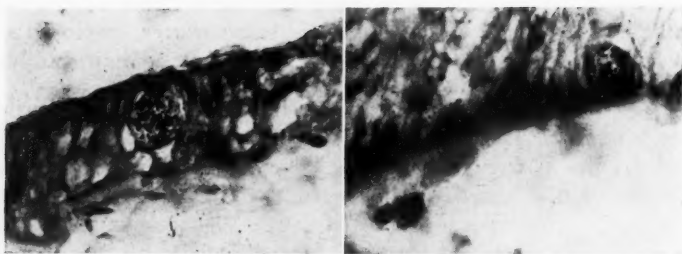


Fig. 1.—A large "vacuole" in the epithelium of the endolymphatic duct. Hematoxylin-eosin. X 950. Note compression or dislocation of the epithelial nuclei.

Fig. 2.—Two "vacuoles;" one intraepithelial, the other lying free in the lumen. Hematoxylin-eosin. X 800.

this (high) epithelium and also on the surface border, as well as in the lumen. The picture of the compression and displacement of the lumenally placed nuclei shows that the vacuoles seem to move toward the lumen (Figs. 1 and 2). Vacuoles may be seen, some of which are free in the lumen while the rest are still embedded in the epithelium (Fig. 3). Moreover, granules of quite the same appearance may be demonstrated in the gelatinous macular substance of the sacculus below the endolymphatic duct and in the utricular recess. The last-mentioned granules differ from the others only in their ground substance being calcified. As particular details in this observation the following data are mentioned:

In the adult and fetal shark, the lining epithelium of the endolymphatic duct consists in the juxtasaccular half of the duct of an unusually high, single, layer of columnar epithelium with oval nuclei and a finely granular cytoplasm, rather light in color. This epithelium varies somewhat in height, forming folds in the lumen, and its cytoplasmic basal zone is characterized by a reticular structure made conspicuous by most cytoplasm stains. The above-mentioned vacuoles are situated in this epithelium, but only in the proximal half of the endolymphatic duct, and they may also be seen in the medial wall of the sacculus below the site of departure of the duct. They are most frequent just distally to the site of the cranial perforation of the duct where as many as eight vacuoles per cross-section may be seen.

The possibility of artefacts should always be kept in mind. In this case, however, it is difficult to imagine that such sharply defined and uniform structures that are present only in the walls of the

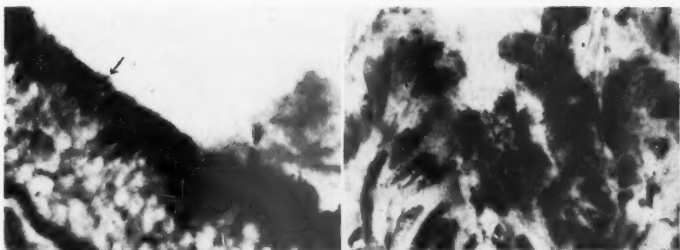


Fig. 3.—A "vacuole" penetrating the surface of the epithelium. To the right a shadow—as is seen frequently in sections of the ductus—possibly pointing at a production of mucus by the epithelium here. Hematoxylin-eosin. X 800.

Fig. 4.—Cross-section of the folded epithelium of the duct presenting a "vacuole." Hematoxylin-eosin. X 900.

duct—and are also seen in micro-incinerated preparations of unstained sections—might have arisen in the course of preparation. Their real intravital existence can be verified if fresh ducts are extirpated and opened and then studied under the microscope with strong side illumination. In such cases the surface of the epithelial lining may be seen to be slightly irregular, and in successful preparations small formations of the size of the described vacuoles can be made out in the proximal half of the duct.

Crystal-optical examination of the vacuoles has been carried out by Hans Clausen, M.Sc., lecturer at the University of Copenhagen (Institute of Mineralogy and Geology) to whom I am greatly indebted for his thorough and expert assistance. From his reports the following data may be cited: "The vacuoles were examined in sections of $10\ \mu$ and stained with hematoxylin dyes. They are found to be sharply defined, spherical formations measuring $20\text{--}60\ \mu$ in diameter and they appear to be separated from the surrounding tissue by a sharp and thin borderline. Their contents are not homogeneous, appearing to be made up of small granules, the exact size of which cannot be estimated readily, but they are about $1\ \mu$ in diameter, and the granules are not equal in size . . ."

The same picture is obtained on examination of the same vacuoles under the phase-contrast microscope, and under the usual microscope as well as by polarization microscopy.

"The birefringency observed in the polarization microscope is so pronounced that it cannot be caused by quartz (see below). From the dimensions of the granules and the interference color they are

taken to be composed of calcium carbonate. The small granules in the vacuoles are double-refractive and are embedded in an optical isotropic ground substance" (from a description of a section from the ductus endolymphaticus).

Concerning one of these sections picked out at random, Clausen says: "... in one of the vacuoles a granule is seen which is of about the same size as that of the nuclei in the cells of the specimen. The granule is several times longer than it is broad, and it shows with crossed nicols a distinct yellow interference color of the first order." The writer has submitted such sections to micro-incineration in which all organic tissue was combusted, and only the inorganic matter remained. The study of such sections showed the presence of vacuoles of quite the same size and distribution, localized as described above. Here, too, the contents of the vacuoles are ascertained to be non-homogeneous and granular, whereas more particular details could not be made out.

When these vacuoles are found at any level in the epithelium and also are seen to be breaking through the surface of the epithelium, it is only reasonable to assume that the granules migrate, and the direction of their migration is elucidated by studying the localization and appearance of the nuclei in the cells compressed and dislocated in front of the vacuoles.

The vacuoles migrate towards the lumen of the duct, and some of them may be seen to project through the epithelium surface into the lumen. They are also seen within the lumen of the duct and, what is very important, they may also be found in the saccular macula-jelly proper (otolithic membrane covering the macula sacculi) as will be mentioned below.

Concerning this point Clausen says: "... and in sections the sacculus was found to contain, among calcite and quartz granules at the place of the otolithic membrane, also grains showing a striking resemblance to the vacuoles described above. The dimension of a typical grain is about $65\ \mu$ and, like the vacuoles, it is seen to be built up of small birefringent particles, which, on an average, are a little larger than the corresponding particles in the vacuoles of the duct. A few of these particles measure up to $6-7\ \mu$ in length but considerably less in width. The dimension of these particles, in connection with the interference color observed, suggests that quartz may here be left out of consideration. By its shape, i.e. its definition against the lumen and its content of birefringent substance in the form of "granules," this grain reminds very strongly of the aforementioned vacuoles seen in the duct. Many grains of this kind

are observed. . . . " It is an interesting fact that the ground substance uniting the granules in the saccular "vacuoles" has become birefringent, whereas the corresponding ground substance in the vacuoles of the endolymphatic duct shows no birefringency.

Accordingly, there can hardly be any doubt that the vacuoles here observed are expelled into the lumen of the endolymphatic duct, from where they pass down into the saccular otolithic membrane. In this connection it is to be mentioned that similar "vacuoles" will also be found in the recessus utriculi.

It is further significant that such vacuoles have been observed nowhere in serial sections from the endolymphatic duct in fetuses, regardless of their size and age. So they must be formed after the birth of the animal. As these vacuoles, moreover, are encountered even in large sharks (over 2 kilo) they must be assumed to be formed throughout life.

EXOGENOUS OTOLITHS

Even at his first examination of the shark labyrinth the writer wondered at the small granules in the large saccular otolithic membrane differing widely in appearance and even in color. The granules reminded him greatly of grains of sand at the waters edge of the seashore.

The following technique was employed for investigation of the nature and origin of these otoliths.

The sacculus was extirpated from a cranium already freed from its soft parts, and the saccular content was scraped down into a small test tube to which was added a little water and then the tube was centrifuged. Thus the contents of the tube were divided: the heavier granules below, the gelatinous substance in the middle and the water above. As far as practicable, the jelly was removed by means of fine pincers and the rest was sent to Professor Tovborg Jensen, the Royal Veterinary and Agricultural College, Copenhagen, for roentgenographic examination. The author is greatly indebted to professor Tovborg Jensen for his help.

A similar tube (with contents from another shark) was sent to Hans Clausen for crystallographic examination.

On roentgenographic examination the fundamental crystalline structure was found to correspond to the structure characteristic of calcite. This applies to 80-90% of the material, while the rest showed lines corresponding to the structure of quartz.

As any formation of excretion of quartz in the shark labyrinth in vivo is most improbable, it must have entered the labyrinth from the outside. (Quartz grains are never found in the labyrinth of *acanthias fetus*.)

The outcome of the crystal-optical examination is quite in keeping with the above, as Clausen says " . . . through microscopic examination it is ascertained that grains of quartz as well as grains of calcite are present. The size of these grains is about 60-70 μ , but several are somewhat smaller, while a few are larger. This applies to the quartz as well as to the calcite grains. A few grains of quartz are observed with dimensions of 200-300 μ .

In the konoscope both calcite and quartz grains may give axial pictures, and suitably oriented grains may be seen optically to be uniaxial-negative and uniaxial-positive, respectively. As to the quantitative proportion of quartz and calcite grains, microscopic examination shows the number of quartz grains to make about 10% of the total number of grains. No grains of any kind of crystals other than the above mentioned (including the "vacuolar grains") have been noticed.

Abundant amounts of small grains are found throughout the endolymphatic duct. Thus, as stated by Retzius, it is possible by pressure upon the area around the outlet of the endolymphatic duct to squeeze out some "otolith mush," and it seems only reasonable to assume that the sharks take up grains of sand through their endolymphatic ducts which open freely on the skin surface over the dorsum cranii.

SUMMARY

It is pointed out that in *Acanthias vulgaris* the otolithic granules are of diverse origin.

They may be formed by the animal itself, either at the site of the macularium or in the epithelial lining of the endolymphatic duct. In the latter place crystal-containing "vacuoles" are formed, which here have been examined in various ways e.g. roentgenographically and crystal-optically. These vacuoles seem to contain calcium carbonate and apparently they migrate out of, or are discharged from, the epithelium into the lumen of the duct. Then they pass down through the lumen to the macular gelatinous substance, in which they become embedded. This process takes place only after the birth of the animal, and presumably it continues throughout life.

In addition, exogenous otoliths are encountered in the form of grains of sand taken up through the patent ductus endolymphaticus and likewise embedded in the macular gelatinous substance.

It is a striking fact that the endogenous otoliths, constituting about 90% of the total otolith mass, are made up of calcite, whereas the endogenous otolithic granules found in all other animal species examined consist of aragonite, the other crystalline form of calcium carbonate.

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UPLANDSGADE 36 B.

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Clinical Notes

LXXXV

LIPOMA OF THE LARYNX

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BETHESDA, MD.

The first case of lipoma of the larynx was described in 1854.¹ Since then, there have been references to some 32 cases in the literature.² Chevalier Jackson has seen seven cases in 40 years.

According to location, they are classified as intrinsic or extrinsic. The former lie in the vestibule, ventricular band or ventricle. There has been no reference to cordal or subglottic origins. The more common extrinsic origins are the anterior surface of the epiglottis, valleculae and aryepiglottic folds. They may also originate from the posterior aspect of the larynx and pyriform sinus. Often they are pedunculated and may thus produce intermittent dysphagia or dyspnea. In the original case reported, death by asphyxia was conjectured because no other cause was found at autopsy.¹ The thyroid cartilage may be thinned,³ and the growth may perforate the thyrohyoid membrane and present subcutaneously.⁴

Only five cases of lipoma of intrinsic origin have been noted in the literature.⁴ Of these, one is described as presenting in the left ventricular band region but also filling the left vallecula and extending through the thyrohyoid membrane subcutaneously. It seems quite possible that this could have been an extrinsic growth with the extension into the ventricular band region in the interval between the epiglottic and thyroid cartilages. In intrinsic lesions there may be hoarseness, stridor and hoarse cough. These symptoms are absent in extrinsic lesions. A muffled voice and intermittent dysphagia and dyspnea characterize the extrinsic type. The age incidence of lipoma of the larynx has varied from four to 83,^{2, 4, 9} The tumor is usually pink with yellowish areas suggestive of cyst formation and this has led to exploratory puncture in several reported cases.⁴ Histologically, these tissues are covered by intact epithelium, usually squamous in type, and the stroma resembles adult fatty tissue. Theoretically these tumors may arise from:

1. Existing fat cells which occasionally occur in small clusters in the walls of the larynx.
2. Embryonic rests.
3. Fibrous connective tissue which still has multiple potentialities.

While lipomata usually occur in fatty sites, they are also found in areas where fat is normally absent, eg. gut, lung and pleura.⁴ Other ideas that have been mentioned are that these tumors are fibromata which undergo fatty degeneration⁶ or that, due to altered local chemistry, fat is either absorbed or accumulates because of failure of catabolism.⁵

Sometimes these tumors are associated with myxomatous or cartilaginous tissue,^{2, 8, 9} and they are then given hyphenated names such as chondro-myxo-lipoma.

Treatment has been varied. Pedunculated growths have been successfully removed by hot and cold snare under direct or suspension laryngoscopy. When this method was tried on broad based lesions, multiple operations were required, and recurrence was the rule. A common finding was that when a presenting tumor was removed, there were more deeply situated tumors which then came into view. Several lesions were removed by subhyoid pharyngotomy and one by incising the cheek from the angle of the mouth to the masseter muscle. Laryngofissure is advocated for intrinsic tumors and tracheotomy may be necessary for dyspnea or as an adjunct to endolaryngeal treatment.¹⁰ On the recommendation of Dr. Clerf, we approached the tumor via the transhyoid route.

CASE REPORT

E. L. S., a 56 year old male was operated upon at Hahnemann Hospital for a grade three adenocarcinoma of the cecum in December, 1948. A side to side ileo-transverse colostomy with resection of the right colon was done. On January 23, 1950, he was admitted to the U. S. Naval Hospital, Philadelphia, Pa., for treatment of a recurrence in the incision noted since November, 1949. On February 1, 1950, the abdominal wall tumor was excised and the wound closed with the aid of tantalum mesh and steel wire sutures. The pathological report was grade two adenocarcinoma invading most of the tissue submitted for examination. After operation, he developed signs of increasing intestinal obstruction. On March 16, 1950, he was reoperated upon for a recurrence involving the pelvis and small bowel. The pelvic mass was excised and a loop of small gut was resected. Stones were noted in the gall bladder. The anesthet-



Fig. 1.—Preoperative mirror view of larynx.

Fig. 2.—Postoperative mirror view of larynx.

ist noted difficulty in intubation due to a "polyp." On April 14, 1950, he was referred for laryngoscopic examination. Indirect laryngoscopy showed a large pink and yellow mass filling the valleculae and pushing the epiglottis postero-inferiorly. The patient stated that ten years ago he experienced a "fluttering" sensation in the throat. A mass was removed with a snare and the base cauterized with phenol at that time. About eight months ago he noted vague sensations in the vicinity of the larynx. During the past three months, he noticed a change in his voice with a sensation of talking with a foreign body in his mouth. When lying on his back, he noted a "fluttering" in his throat. There was no dyspnea and the voice was muffled but not hoarse. On April 20, 1950, a biopsy was performed under direct laryngoscopy. Up to this time, cyst and lipoma were considered in the differential diagnosis. Biopsy report was lipoma. A transhyoid pharyngotomy was performed under endotracheal anesthesia on April 27, 1950. Through a transverse incision over the hyoid bone, the hyoid bone was severed in the midline with a gigli saw. The thyrohyoid membrane was incised vertically and normal fat in the preepiglottic space was pushed aside to expose a large well demarcated tumor deep to the valleculae. The second tumor was exposed with more difficulty because it was attached to the anterior surface of the epiglottis. The tumors were about four cm and two and one-half cm in diameter. The mucous membrane was closed with catgut and the hyoid bone was brought together by suturing the peristernum and ribbon muscles. A penrose drain was inserted for 48 hours.

Postoperative course was uneventful except for a temperature rise to 100 degrees on the first postoperative day. Skin sutures were removed on the sixth postoperative day when the wound was healed.

The pathological report on the excised specimen by Dr. B. K. Black is as follows:

Microscopic study of numerous sections taken from the tumor show it to be composed primarily of large lobules of normal appearing fat cells. These lobules are separated by wide bands of edematous connective tissue. Within the connective tissue there are islands and strands of irregularly arranged spindle shaped cells, each with a long narrow nucleus and a moderate amount of eosin-staining cytoplasm. There is moderate pleomorphism but no bizarre mitotic figures are seen.

There are no areas of palisading or of mucoid degeneration. These cells closely resemble the characteristic cell of the neurofibroma.

Sections which include the surface of the tumor show the submucosa blood vessels to be markedly dilated. There is a slight to moderate diffuse infiltration of the submucosa with lymphocytes and a few plasma cells. The overlying mucosa has undergone squamous metaplasia. The tumor does not involve the mucosa.

Diagnosis: Lipoma containing neurogenic elements.

He has no further difficulty with his larynx, but is running the usual adverse course of recurrent carcinoma of the bowel.

SUMMARY

A case of recurrent lipoma of the larynx associated with adenocarcinoma of the large bowel is reported. The literature on lipoma of the larynx is reviewed.

U. S. NAVAL HOSPITAL.

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ADENOMA OF THE CERUMINOUS GLANDS

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AND

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Adenoma of the ceruminous glands is an extremely rare tumor involving the external auditory canal. An excellent review of the literature and a report of the third proven case of this type of neoplasm was presented by Adler and Somner¹ in 1944. The following case presents much of the same symptomatology as the one which they reported.

A 27 year old colored female was seen June 26, 1951, in the outpatient clinic of the Department of Otolaryngology at the Medical College of Alabama complaining of gradually increasing deafness in the left ear during the preceding 18 months. The hearing loss was accompanied by occasional pain in the ear for six months. A week prior to examination the pain became intense and constant. A member of her family had noticed a "growth" in the patient's left external auditory canal several days prior to the time she sought medical attention. The patient did not complain of tinnitus or vertigo.

Examination of the left ear revealed a bluish gray, fibrous, encapsulated tumor which completely filled the external auditory canal and protruded four to six mm therefrom. It appeared to be attached to the postero-superior surface of the canal, caused pain on manipulation, was freely movable, and did not appear to be infiltrating the surrounding tissue. Cervical glands were nonpalpable; mastoid x-rays showed no bone destruction. Audiometric study revealed a 60 decibel loss at 2000 and 3000 cycles.

The patient was admitted to the hospital where under sodium pentothal anesthesia an endaural incision was made and the auricle retracted. The skin of the posterior, inferior, and superior portions of the canal was elevated and the neoplasm was found to be arising

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from the skin of the postero-superior portion of the external auditory canal. There was no invasion of the bony wall of the tympanic membrane. The canal was packed lightly with sea sponge and one suture taken in the temporal extension of the incision.

The tissue removed was tubular in shape, 2.3 cm in length, and up to 8 mm in diameter. Multiple cross sections revealed no lumen.

Microscopic appearance as given by Dr. Joseph Cunningham was as follows: "Epithelial cells grow in cords. Some glands spaces are present with a little pink staining material in the lumina. In other places there are vessels in between. There is some resemblance to the epithelium lining sweat glands. Comments: The tumor suggests one which has arisen from appendages of the skin. There are some features like those of basal cell carcinoma though there seem to be definite lumina here which is not usual in the basal cell carcinomata. Origin from sweat glands seems less likely because there are said to be no sweat glands in the external ear canal. An origin from ceruminous glands is to be considered. Mitoses are not at all conspicuous and the cells are fairly uniform."

The patient was examined repeatedly in the clinic for approximately five weeks postoperatively, at which time the operative site had epithelialized completely. Postoperative audiogram revealed a 20 decibel improvement in hearing.

CONCLUSION

A clinically benign tumor removed from the skin of the external auditory canal is presented with the pathological diagnosis of ceruminous gland adenoma.

MEDICAL COLLEGE OF ALABAMA.

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LXXXVII

ENDONASAL SCHWANNOMA

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AND

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The case which is presented here corresponds to a very unusual nasal tumor, of which there are but a few examples in the world's medical literature.

Masson's schwannomata were so called because of their origin in Schwann cells. The designations of neurinoma (Verocay) and neurilemmoma, are also applied to this tumor. Ribbert places it among fibromata (neurofibromata); Ewing among the tumors of the peripheral nerves. Aschoff differentiates the real neuroma-proliferation of nerve cells from neurofibromata, and places the neurinoma intermediately between the two.

It is known that nerves are made up of nerve fibres, which, in turn, are formed of a group of neurofibrils with their layers of myelin and their covering neurilemmas. The neurilemma is formed of Schwann cells, the pathological proliferation of which would give origin to neurinomata.

According to Ewing, neurinomata are nerve tumors of a most characteristic type; Babini and his collaborators consider them the most frequent tumors of peripheral nerves. The most frequent locations would be the spinal cord, the acoustic nerve (the tumors of the cerebellopontine angle, are often schwannomata), the stomach and small intestines.

From our search of the literature between 1936 and 1946, we list the following locations in the order of their frequency: stomach, acoustic nerve, thorax, intestine and peripheral nerves.

The tumors are of slow growth, and may become very large, although in this respect we must take into consideration that once they have reached a certain size they remain stationary and even retrogress by necrobiotic involutionary processes.

Symptoms are due to the compression of the adjacent organs. However, they may present varying degrees of malignancy and act as a malignant tumor, giving the same symptoms.

The tumor must be radically removed, and in general, there are no recurrences.

As with every other nerve tumor, this one is, on the whole, insensitive to x-rays. In the bibliography we found four cases of orbital neurinomata treated, two of them with radium and two with x-rays, without result; but an American author, not available to us, has reported a five year cure with x-rays.

Not more than five cases of nasal schwannoma are found in the world's literature: four in German literature, with origin in the ethmoidal sinuses, in the maxillary sinuses and in the middle meatus; and two in American literature, one of the maxillary sinus and another of the front part of the nasal septum. Cruveilhier cites a case in which the tumor came out of the nose, but did not originate in it, but in the second branch of the trigeminal nerve.

In our case the tumor comes from the region of the cribriform plate, although it is not a schwannoma of the olfactory nerve, as the first pair lacks a neurilemma and therefore Schwann cells. Probably it originated in a small branch of the anterior ethmoidal nerve.

More common are nasal gliomata, and it is important to make a differential diagnosis, because these tumors present themselves almost exclusive in childhood, while schwannomata appear at any age (two to 70 years). Gliomata are quite sensitive to radium. The differentiation is not difficult because the histopathological picture of the neurinoma is quite characteristic, with its nuclei "in palisade."

REPORT OF A CASE

Mr. S. L. T., Italian, aged 40, married, working as a day-laborer, came to our service on May 16th, stating that he had been suffering from this disease for the past three months. It began with anosmia and then nasal obstruction and mucous secretion, symptoms which he neglected, attributing them to a cold.

Afterwards appeared a swelling in the right nasal fossa which grew until it appeared at the naris. It was of a wine-red color, of a fleshy consistency, superficially ulcerated, exerting pressure upon the nasal walls and bleeding at the least contact. Biopsy revealed the following: In the histological preparation may be observed a tissue of well ordered fasciculated structure, formed by cells with elongated nuclei and slightly fibrillar cytoplasm. In some fields the nuclei are

arranged in palisades or whorls. In some, recent hemorrhagic foci are observed. No characteristics of malignancy are seen, but cell abundance renders it necessary to make a reserved prognosis. Diagnosis: fasciculated Schwannoma (Cid).

The radiographic examination showed opacity of the right maxillary and fronto ethmoidal sinuses. Personal records showed syphilis, acquired at 32, treated with injections for three years, after which the patient was discharged, apparently cured.

He had lived in Ethiopia for ten years, from 1939 to 1949. He had had also typhoid and gonorrhea.

Under local anesthesia a rhinotomy was done following the fronto-naso genian sulcus. An enormous reddish-gray swelling of even surface appeared, bleeding, which occupied both nasal fossae and prolonged itself towards the fronto-ethmoidal zone. The nasal bones had become considerably thinner, and were easily extirpated with the Luc forceps. The ascending branch of the upper maxillary and the unguis were resected with previous separation of the lachrymal sac. Both frontal sinuses were opened. They were covered with granulations and tissue of a polypous appearance. It was necessary to widen the operative opening, and the anterior wall of the sinuses and the frontal spine were removed for this purpose. This allowed good access toward the seat of the tumor which was at the same height as the cribriform plate.

A thick curette was introduced on a level with the floor of the nasal fossa, and insinuating it behind the tumor firm but soft pressure was exerted with a swinging movement and the tumor removed.

Hemorrhage was stopped by pressure. The nasal fossa was filled with iodoform gauze. The cutaneous wound was closed with silk. A blood transfusion of 250 cc penicillin and calcium was given. On the fifth day the cutaneous stitches and half the gauze were taken away. Three days later the rest of the gauze was withdrawn. During the following day there welled up a scanty serosanguineous secretion. The nasal fossa was wide, permeable and even.

He was discharged completely cured and a month later appeared to be perfectly well. Radiological examination showed the facial sinuses clear and free.

FACULTAD DE CIENCIAS MEDICAS.

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THE AWARD OF MERIT

The Award of Merit of the American Otological Society for 1951 has been given to

BARRY G. ANSON, PH.D.

Professor of Anatomy, Northwestern University,
School of Medicine
Chicago, Illinois

and

THEODORE BAST, PH.D.

Professor of Anatomy, Wisconsin School of Medicine,
Madison, Wisconsin

for their joint research contributing to the knowledge of "The Embryology in Anatomy of the Temporal Bone and the Ear." The Award of Merit Medal is accompanied by an honorarium and is granted by the American Otological Society, Inc., for the purpose of recognizing outstanding service in research in otology and in the interest of encouraging research in the problems of impaired hearing.

The Scientific Papers of the American Otological Society

LXXXVIII

SOME ANIMAL EXPERIMENTAL FINDINGS ON THE FUNCTIONS OF THE INNER EAR

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ZÜRICH, SWITZERLAND

I should like to discuss today the results of several experiments performed on guinea-pigs in regard to certain metabolic processes taking place in the inner ear.

Ever since Schulze traced the fibers of the auditory nerve to the organ of Corti, and Retzius and Held demonstrated the further course of these fibers to the hair-cells, these hair cells generally have been considered the chief sensory elements in which the transformation of mechanical sound energy into nervous excitation is said to take place. Occurrence of electrical potentials within the cochlea, among other things, is proof of this process whose essential points are still unknown. The following observations show that these cochlear microphonics, the Wever-Bray effect, are due to the stimulation of the hair cells. First of all, in certain animals with congenital deafness, for instance in the albino cat, the dancing mice, the waltzing guinea pigs and a strain of Dalmatian dogs, severe histological changes, or even total loss of the organ of Corti can always be found. On these animals the Wever-Bray effect cannot be demonstrated. Secondly, in several animal experiments the auditory nerve has been severed, resulting in degeneration of the descending nerve fibers and ganglion cells of the cochlea. In those animals which show a normal organ of Corti, in spite of the descending degeneration, normal cochlear microphonics in response to sounds can be determined. Thirdly, it is possible to injure experimentally, for instance by overstimulation, the area of the hair cells, thus causing a decrease of cochlear microphonics.

First we shall take a closer look at the morphological changes in the organ of Corti caused by overstimulation, and compare them with the lesions in the inner ear due to intoxication with streptomycin

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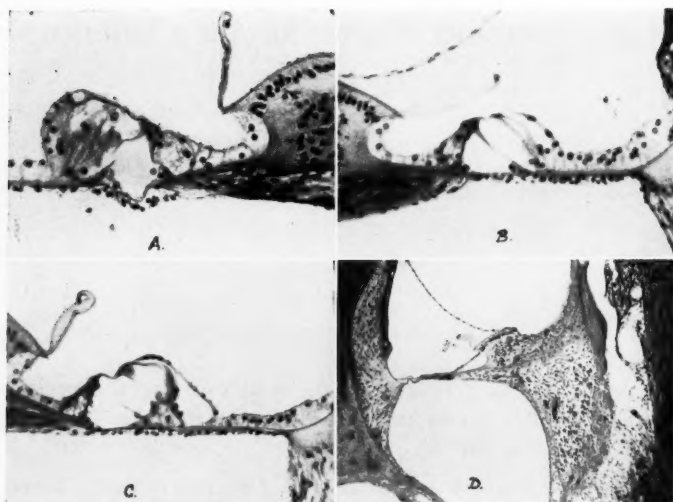


Fig. 1.—(A) Chromatolysis in the nuclei of the outer hair cells one hour after exposure to 12 reports of a 7.5 mm caliber revolver at a distance of 50 cm. (B) Loss of the outer hair cells after exposure to one report of an anti-aircraft gun of 2 cm caliber at a distance of 2 meters. Recovery period of 65 days. (C) Loss of the outer hair cells after exposure to synthetic noise (1750 to 10,000 c.p.s.). Recovery period of 280 days. (D) Disintegration of the organ of Corti after exposure to 12 reports of an anti-aircraft gun of 2 cm caliber at 2 meters distance. Recovery period of 67 days.

and quinine. Pure tones, noises and gun reports produce in the main the same changes in the organ of Corti in proportion to sound energy and exposure time. Depending, however, on whether they are caused by pure tones or noises, these changes show entirely different, partly unsystematic locations. Immediately following a short time (less than one hour) of exposure to pure tones (1000-7000 c.p.s., with a sound intensity of 135 db), as well as immediately following exposure to gun reports, no histological alterations as a rule could be found in our animals. Specifically, the severance of continuity in the organ of Corti, following high sound pressure, and a stripping of the tympanic lamella as observed by M. H. Lurie and Kendon R. Smith could not be found. Only in exceptional cases, following the introduction of pure tones of 4500 c.p.s., at 140 db sound pres-

sure did we find hemorrhages in the scalae of the cochlea and rupture of the membrane of Reissner, as described by Hallowell Davis and his co-workers. But as early as one hour after exposure to 12 reports of a 7.5 mm revolver from a distance of 50 cm (Fig. 1A) the first typical damages could be seen in the sensory cells of our guinea pigs. The cell nuclei of the two median outer hair cells were bullous and enlarged, the chromatin was conglobated into small balls. Soon thereafter these most likely dead cell nuclei shrank. The cytoplasm bulged out of the walls of the cells, thus making it impossible to recognize clearly the boundaries of the cells. A few hours later the Deiters' cells also underwent a similar process of degeneration. Sixty five days after exposure to one report of an anti-aircraft gun of 2 cm caliber at a distance of 2 meters the frequently split outer pillar cells on one hand (Fig. 1B), and on the other hand, the most lateral cells of Deiters and Hensen which remained intact for a long time formed the walls of a cavity, the roof of which was formed by the lamina. The space of Nuel and the external tunnel were enclosed in it. This concluded the first stage of the histological cell damage, which was characterized by a circumscript loss of the outer hair cells. No further pathological changes could be found at this stage in the other parts of the organ of Corti. This circumscript loss of outer hair cells in guinea pigs (Fig. 1C) exposed to synthetic noise of 1750 to 10,000 c.p.s., at 100 db sound pressure over a period of 2347 hours remained unaltered even after a recovery period of 250 to 280 days. Consequently a loss of hair cells, once it has occurred, must be considered permanent. In the zone of inquiry of the outer hair cells the finest nerve fibers are also destroyed, but at this first stage of the degenerative process no retrograde degeneration of the nervous elements could be found as yet.

Loss of the internal hair cells marked the beginning of the second stage of the damage to the sensory cells (Fig. 1D). Simultaneously all support cells of the organ of Corti dissolved. The whole architecture collapsed rapidly and disintegrated. In place of the organ of Corti only a thin layer of cubic cells sat on the basilar membrane, while the cells of the tympanic lamella in irregular order adhered to the membrane, some with flattened cell bodies, others with shrunken nuclei.

The second stage of the process of degeneration, characterized by destruction of the organ of Corti, may take a very rapid course.



Fig. 2.—Disintegration of the organ of Corti after exposure to 4500 c.p.s. at an energy level of 140 db over 38 hours. The beginning of retrograde degeneration of nerve fibers and cells may be seen in this section.

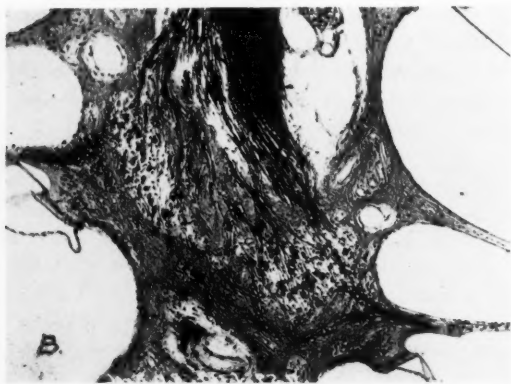


Fig. 3.—Retrograde degeneration of the cochlear ganglion and of the centripetal nerve fibers going to the eighth cranial nerve.

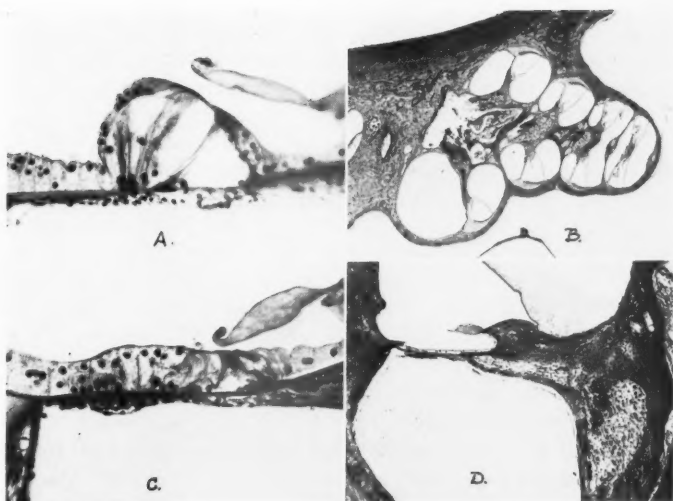


Fig. 4.—(A) Loss of the outer hair cells in the first turn of the cochlea following streptomycin administration. (B) Disintegration of the organ of Corti with retrograde degeneration of the nerve apparatus following streptomycin administration. (C) Loss of outer hair cells in the first turn of the cochlea following the administration of quinine. (D) Disintegration of the organ of Corti, with retrograde degeneration of the nerve elements of the first turn of the cochlea following the administration of quinine.

Overstimulation with 4500 c.p.s., at 140 db, over as little as 38 hours, may lead to complete loss of the papilla auditiva. Following exposure to 12 reports of a 7.5 mm revolver from a distance of 50 cm, the organ of Corti disappeared within seven days.

After complete destruction of the organ of Corti, the corresponding nervous apparatus also showed signs of retrograde degeneration. Thus we found (Fig. 2) after 38 hours of overstimulation with a pure tone of 4500 c.p.s. in the nerve channel of the lamina spiralis ossea a loose plexus of varicose nerves with poorly stainable myelin sheaths in place of the densely interwoven bundle of medullated nerve fibers. As the degenerative process progressed (Fig. 3), fat granule cells and pigment granule cells showed up between the degenerating nerve fibers. Soon the corresponding ganglion spirale showed damage. Some ganglion cells lost their Nissl bodies and the

nucleus was displaced toward the cell periphery. Some ganglion cells finally dissolved. The ganglion spirale appeared thinned out due to the loss of cells. Signs of degeneration were also found in the nerve fibers ascending from the ganglion to the auditory nerve.

Now we will proceed to consider briefly the histological changes in the inner ear of the guinea pig due to streptomycin and quinine poisoning, and compare them with the degeneration following overstimulation.

The excellent French scientist R. Caussé, who unfortunately died far too soon, was the first to notice histological changes in the peripheral vestibular apparatus produced by streptomycin. He observed such changes in three mice poisoned with streptomycin. In one of these mice, furthermore, he found degenerative changes in the first cochlear turn of the organ of Corti. The absence of outer and inner hair cells was accompanied by a degeneration of the corresponding nervous elements. K. Berg reported similar changes in the peripheral auditory organ of cats poisoned with streptomycin. A short time ago we were able to prove that in the guinea pig, too, protracted injections of large doses of streptomycin will produce severe damage in the peripheral vestibular apparatus and within the cochlea (Fig. 4A). Here the outer hair cells were the first to disappear. In the second stage (Fig. 4B) rapid disintegration of the organ of Corti set in with the loss of the inner hair cells, accompanied by a retrograde ascending degeneration of the nervous elements in the area of the first turn which was damaged most severely.

Regarding the effects of quinine on the ear, Covell has been able to show that the degenerative process histologically also begins in the outer hair cells. In agreement with his findings, by protracted administration of large doses of hydrochloric quinine we have been able to produce (Fig. 4C) a loss of outer hair cells over wide areas of the cochlea in guinea pigs, too. In the following stage (Fig. 4D) of degeneration quinine, too, caused disappearance of the organ of Corti with retrograde degeneration of the nervous elements, chiefly in the area of the first turn.

According to these observations the histological changes in the organ of Corti caused by streptomycin and quinine correspond to a large extent with the degenerations due to overstimulation.

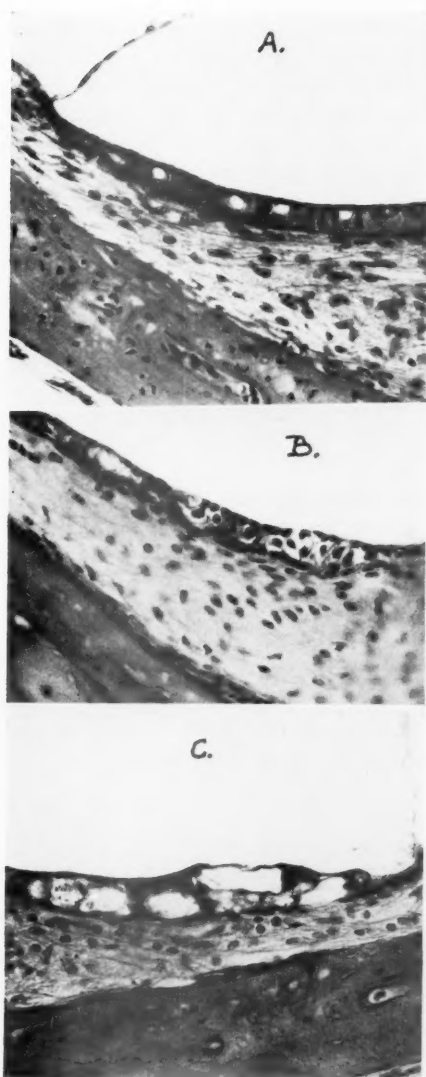


Fig. 5.—(A) The normal stria vascularis of the guinea pig. (B) The stria vascularis in the area of disintegration of the organ of Corti due to streptomycin. Disruption of the surface epithelium by dilated capillaries may be seen. (C) The stria vascularis in the area of disintegration of the organ of Corti due to quinine. Marked dilatation of the capillaries is present. Disruption of the surface epithelium and decrease of epithelial elements may be detected.

In the streptomycin and quinine poisoned guinea pigs, however, additional marked changes occurred in the morphological structure of the stria vascularis. The normal surface of the stria (Fig. 5A) is lined by a continuous layer of cubic epithelial cells whose nuclei are lined up in a single column near the surface. In guinea pigs poisoned with streptomycin (Fig. 5B) and quinine this epithelial lining was disrupted in several places throughout the area of those turns in which the organ of Corti had disintegrated. In the epithelial defects dilated capillaries of the middle layer of the stria were found. There the endothelium of vessels having no connective tissue was in direct contact with the endolymph. In the advanced stage of poisoning (Fig. 5C) a part of the vessels of the stria showed cystic dilatations, while there was a marked decrease of epithelial elements between the vessels. Thus the thickness of the stria vascularis, which was swollen initially, decreased.

Based on the histological changes in the inner ear, one gets a very clear insight into the mechanism of sound injuries. According to the findings of the Bostonian group (Hawkins, J. E., Lurie, M. H., and Davis, H.) excessive sound energy, conducted to the organ of Corti via the oval window and the fluid of the inner ear, produces gross rupture of the sensory epithelium. Contrary to this view, we are more inclined to assume that the over-loud sound causes invisible damage to the molecular structure of the sensory cells which primarily remain intact. Following this direct trauma to the hair cells, the other degenerative processes develop secondarily. In this process the organ of Corti may disappear completely within 38 hours, which is an extremely short time. M. H. Lurie, who has been working on the problem of degeneration and absorption of the organ of Corti in animals due to overstimulation, stated that the degeneration that takes place in these animals is of the same type as that which occurs naturally in animals with congenital deafness, for example in the waltzing guinea pigs and in the dancing mice. Lurie has injected trypan blue into a number of overstimulated and animals born deaf in order to ascertain if any endothelial cells participate in the process of absorption of the organ of Corti. But no evidence of any such action could be found. In Lurie's opinion certain alterations found in the stria vascularis did not give any clue as to the mechanism of what occurred in the organ of Corti, nor could any signs of increased phagocytosis be noticed in the ductus and saccus endolymphaticus during or after



Fig. 6.—The effect on the organ of Corti of overstimulation with a pure tone of 4500 c.p.s. at an energy of 140 db over a 38-hour period. In the area of disintegration of the organ of Corti, the stria vascularis is widened due to dilatation of the capillaries.

disintegration of the organ of Corti. The trackless disappearance of the organ of Corti has remained a puzzle so far.

The conditions prevailing in the mechanism of injury due to streptomycin and quinine are much more complicated. Aside from the venous *vas spirale*, the organ of Corti does not possess any nearby blood-vessels of its own. Consequently, the nutrition and oxygen supply of the sensory cells, as well as any poisoning effect, has to take place via the endolymph in which the organ of Corti swims. It is the general belief that the endolymph is secreted by the stria vascularis. Drugs brought by the blood stream also are taken into the endolymph via the stria vascularis. It is interesting to note that in the area of destruction of the organ of Corti guinea pigs poisoned with streptomycin and quinine dilatation of the blood vessels and a decrease of epithelial elements of the stria vascularis are constantly found. Consequently couldn't it be possible that these changes in the stria occur during the transmigration of the drugs from the blood into the endolymph? In this case the damage to the organ of Corti might only be secondary, due to nutritional disturbances.

The following observations speak against the assumption of such a primary damage to the stria vascularis.

The dilatations of the vessels of the stria vascularis occur only in the last phase of the degeneration, accompanied by the disappearance of the organ of Corti.

Such changes in the stria are restricted to the area of those turns in which the organ of Corti disintegrates.

According to all expectations, primary lesions of the stria, planted via the blood stream, should be found equally distributed in all turns of the cochlea. Among the arsenic compounds, there are some which directly affect the stria vascularis. Thus the Japanese authors, T. Miyamoto, H. Nakamura, and J. Ozeni, by the application of atoxyl (sodium arsenilicum) in guinea pigs have produced degenerations of the organ of Corti, of the ascending nerves and of the ganglion cells, which were equally distributed over all turns of the cochlea. In addition to these processes, in the early stages of these experiments, swelling of the stria vascularis occurred in all turns. Later on the entire surface epithelium of the stria degenerated. In control experiments, which we performed on guinea pigs, we confirmed the toxic effect of arsenic, first noticeable in the stria vascularis. As expected, the epithelial lesions occurred in equal distribution in all turns of the cochlea.

Finally, the changes in the stria due to streptomycin and quinine mainly affect the circulatory system, whereas the arsenic poisoning primarily causes degenerative swelling, cloudiness, and disintegration of the epithelial cells.

For the reasons I just mentioned, it is highly improbable that by streptomycin or quinine the stria is damaged primarily. We are more inclined to believe that the drugs, taken into the inner ear by the blood stream, harmlessly pass the stria vascularis to enter the organ of Corti via the endolymph. Then, following resorption which takes place here, the outer hair cells, and later on the inner hair cells and the organ of Corti are affected, too. In this case, the now noticeable dilatation of the blood vessels in the stria would not be a sign of toxic effects, but a response of the secretory organ to the process of disintegration of the organ of Corti.

The coincidence in space and time of the dilatations of the vessels in the stria and of the disintegration of the organ of Corti is all that speaks for this assumption at this moment. If this temporal combination of histological changes corresponds to the functional connection we assume, similar responses of the stria vascularis should be demonstrable in cases of destruction of the organ of Corti due to other causes. Fortunately, the acoustic trauma which, as I have

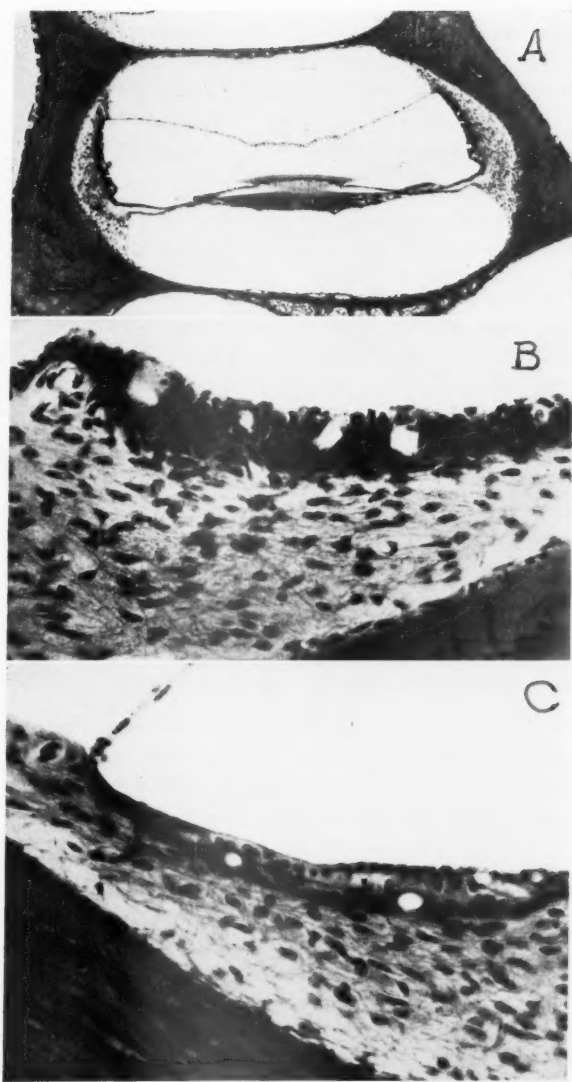


Fig. 7.—(A) Overstimulation with a pure tone of 1600 c.p.s. at an energy level of 140 db for a period of 64 hours. Recovery period, 26 days. In the area of disintegration of the organ of Corti the capillaries of the stria vascularis are dilated and the surface epithelium is disrupted. (B) Enlargement of an area of the stria vascularis shown in (A). (C) Region of the organ of Corti after exposure to 12 reports of an anti-aircraft gun of 2 cm caliber at a distance of 2 meters. Recovery period, 63 days. In place of the surface epithelium, capillary walls line the endolymphatic space in the area of disintegration.

pointed out, attacks the organ of Corti directly, gives us an ideal experimental arrangement for the investigation of this problem. Therefore, we again looked through the histological section of the ears of guinea pigs which had been overstimulated by pure tones, noise and detonations, and obtained the following results:

In the first stage of degeneration of the organ of Corti, characterized by circumscribed loss of the outer hair cells, the stria vascularis showed a normal structure in the affected turns, too.

In the second stage of degeneration, however, characterized by the disintegration of the organ of Corti, marked changes could be found in the stria corresponding to those parts of the turns in which the disintegration of the organ of Corti was taking place or had already been concluded. These new observations can be proved by the following examples:

a) Following pure tone overstimulation (Fig. 6) with 4500 c.p.s., 140 db, over 38 hours, due to extensive blood filling of the dilated capillaries the stria vascularis was markedly thickened in those parts of the second cochlear turn in which the organ of Corti had disintegrated. By this process the cubic cells of the surface epithelium were separated at several points. Thus the epithelium of the blood vessels came into direct contact with the endolymph and the cell detritus deposited on the surface of the stria.

Similarly, seven days after pure tone overstimulation with 7000 c.p.s. at 140 db, over 70 hours, the stria in the area of destruction of the organ of Corti was thickened by dilated capillaries and the covering epithelium was disrupted at various points.

b) Following a 26-day recovery period after pure tone overstimulation (Fig. 7A) with 1600 c.p.s., at 140 db, over 64 hours, vascularisation of the stria and direct contact between vascular endothelium and endolymph at various points could be demonstrated in the area of destruction of the organ of Corti (Fig. 7B). At one point macrophagocytic endothelia seemed to be devouring some cell-detritus.

c) After a seven-day recovery period following exposure to 12 reports of a 7.5 mm revolver from 50 cm distance, dilated capillaries were found in direct contact with the endolymph in the area of the second turn where the organ of Corti had been resorbed.

Secretion and Resorption of Endolymph
(St. R. Guild)

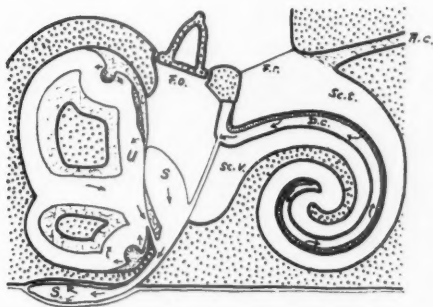


Fig. 8.—The production and absorption of the endolymph according to Guild.

Production of Perilymph

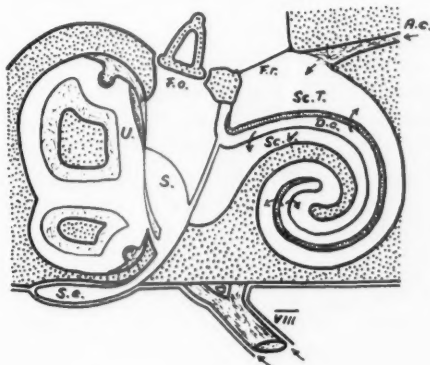


Fig. 9.—The production and absorption of the endolymph according to Saxén and von Fieandt.

After a recovery period of 63 days following exposure to 12 reports (Fig. 7C) of an anti-aircraft gun of 2 cm from a distance of 2 meters, the superficial epithelial lining of the stria in the area of destruction of the organ of Corti was replaced at several points by capillaries running parallel to the surface. In this segment of the stria the epithelial elements between the dilated blood vessels appeared markedly decreased.

Summing up, in the second stage of degeneration following overstimulation by pure tones and gun reports we found changes in the corresponding stria vascularis in the form of dilated blood vessels accompanied by disruption of the surface epithelium at various points, and decrease of the epithelial cells lying between the vessels.

It must be considered unlikely that, with the exception of explosion, overstimulation causes primary damage to the stria vascularis. The circumscribed dilatation of the blood vessels, therefore, would seem to represent a functional response of the stria vascularis to the process of disintegration of the organ of Corti. It seems justifiable to assume that the dilatation of the capillaries and the disruption of the surface epithelium of the stria occurs at all points where the organ of Corti, due to any cause whatever, disintegrates, and where fast removal of cell detritus is necessary.

The assumption that under certain conditions the stria vascularis can carry out resorptive functions also does not conform to the present idea of the circulation of the fluids of the inner ear. According to this idea, the stria vascularis is the secretory organ *par excellence* of the endolymph (Fig. 8). According to the iron salt experiments of Stacy R. Guild and similar experiments of S. I. Doi, the resorption of the endolymph and of foreign bodies and waste-products contained therein, is said to take place in the ductus and saccus endolymphaticus. Recently, however, J. R. Lindsay, after apparent experimental destruction of the saccus and ductus endolymphaticus in the monkey suggested that the maintenance of a normal quantity of endolymph is not dependent on the existence of the endolymphatic sac or the more differentiated medial dilated portion of the ductus. At least the resorption of the endolymph does not appear to take place exclusively in the ductus and saccus endolymphaticus.

According to Arno Saxén and von Fieandt the sulcus spiralis externus (Fig. 9) plays a part in the resorption of endolymph, as

Production and Resorption
of Endolymph
(Saxén and von Fieandt)

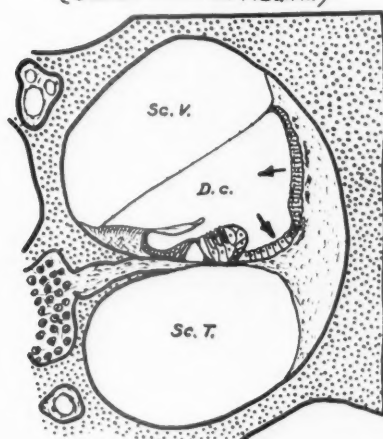


Fig. 10.—Production of perilymph according to Rejtoe and Wittmaack.

"the epithelial cells of the sulcus spiralis externus, provided with long protoplasmic fibrils extending into the connective tissue of the ligamentum spirale, may function as phagocytic groups." Franz Altmann and J. G. Waltner injected iron salt solution into the sub-arachnoid space of rabbits and monkeys, whereafter the spreading of the salt into the inner ear was studied after fixation in formalin-hydrochloric acid solution." Within the cochlear duct resorption of iron salt occurs through the sulcus spiralis externus and the spiral ligament and through the crista spiralis, indicating the resorption of endolymph in the same areas, whereas the stria vascularis is relatively free from Prussian blue granules. The resorptive mechanism of the cochlea seems more than sufficient for the disposal of low molecular substances in physiological concentrations."

Since, based on the permeability of the basilar membrane and the membrane of Reissner, Rejtoe and Wittmaack have assumed that the perilymph (Fig. 10) is derived from the endolymph, the much discussed circulation of the perilymph must also be considered in this

*Production and Resorption
of Perilymph*
(Mygind and Graf)

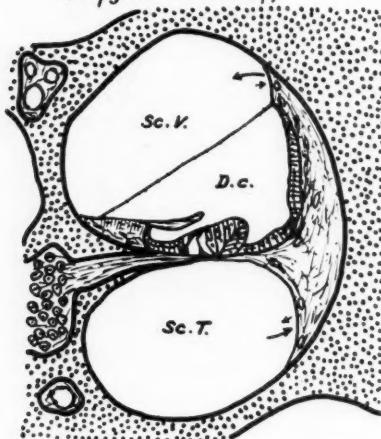


Fig. 11.—Production and resorption of perilymph according to Mygind, Graf, etc.

connection. It is believed by most authors that the perilymph, coming from the cerebrospinal fluid gets into the inner ear through the aquaeductus cochleae, the internal auditory canal and the perineural and perivascular tissue spaces. The presence of erythrocytes in the aquaeduct and along the auditory nerve after brain operations, as well as the demonstration of intracysternally injected Indian ink in the pathways mentioned above, speak for this assumption. Gisselson has been able to observe penetration of cerebrospinal fluid containing fluorescein into the scala tympani via this route. Contrary to this opinion, Aldred, Hallpike and Ledoux consider it unlikely that the perilymph is derived from the cerebrospinal fluid, as they have found that the osmotic pressure in the labyrinthine fluids of the cat is higher than that of the cerebrospinal fluid. Altmann and Waltner, too, in their iron salt experiments could find no fluid current from the subarachnoid space towards the perilymphatic space. The larger portion of the perilymph, therefore, is probably formed in the perilymphatic space itself, most likely by the blood vessels. According to

	macC 45' Tier 179	macC 60' Tier 170	macC 90' Tier 174	macC 120' Tier 172	macC 150' Tier 174
<u>Blut</u>	900	1400	1060	1400	900
<u>KaWa</u>	25	25	50	60	80
<u>Peril.</u>	15	20	30	40	30
<u>Liquor</u>	15	15	25	35	20

Fig. 12.—The concentration of fluorescein in blood, aqueous humor, perilymph and C.S.F. after subcutaneous and intraperitoneal injection of a 10 per cent solution of fluorescein.

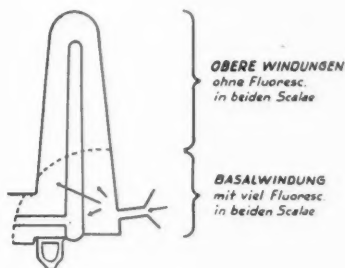


Fig. 13.—The diffusion of fluorescein in the cochlea.

	macC 50' Tier 171B	macC 55' Tier 174B	macC 60' Tier 171A	macC 60' Tier 171B	macC 61' Tier 171A	macC 115' Tier 171B	macC 117' Tier 172A
<u>Blut</u>	5540=98	10000=147	5600=120	13400=158	6100=183	1110=47	6250=129
<u>KaWa</u>	850=28	1210=34	550=39	1620=48	682=25	620=20	1900=49
<u>Liquor</u>	480=21	57,8=25	226=15	710=25	275=11	324=17	1010=43
<u>Peril.</u>	43=96	3,8=98	22=94	5,8=97	26=96	23=94	109=41

Fig. 14.—The concentration of radioactive phosphate in blood, aqueous humor, cerebrospinal fluid and perilymph after subcutaneous injection of an orthophosphate solution in guinea pigs.

Mygind (Fig. 11 and Fig. 17) "a capillary network in the uppermost part of the spiral ligament just above the stria vascularis" ought to be considered the place of origin of the perilymph in the guinea pig. Very little is known about the resorption of perilymph. According to the iron salt experiments of Altmann and Waltner, the resorption of cochlear perilymph seems to take place from both scalae into the tissue spaces and from there into the blood vessels. This route can be demonstrated by injection of India ink into the cistern of guinea pigs.

In experiments on guinea pigs we have been working on the problem of the origin of perilymph and endolymph. In a first series of experiments, my co-worker K. Graf used a ten per cent fluorescein solution for intraperitoneal and subcutaneous injection. Then the fluorescein concentration in the blood, in the aqueous humor of the eye, in the cerebrospinal fluid and in the perilymph was determined by means of a fluorometer. These experiments showed (Fig. 12) that diffusion through the aqueduct can undoubtedly take place, but that the fluorescein is not restricted to diffusion along the pathways of the scalae only, but can penetrate the membranes as well (Fig. 13). Here, too, as in the eye, the question arises whether fluorescein is actually able to give us information about the changes in currents and fluids, or whether it merely reflects the conditions of diffusion. In any case, we must take into consideration the fact that in these fluorescein experiments quite unusual concentrations are employed, leading to gradients which normally do not occur in nature. Since, however, the concentration gradient plays an important part in the diffusion through the aqueduct, the result of the experiments with injection of fluorescein into the subarachnoidal space also cannot be considered proof for natural conditions.

K. Graf, together with G. Poretti, the nuclear physicist, have, therefore, carried out corresponding investigations using radioactive substances, so-called isotopes. Isotopes differ from the ordinary element only in the structure of the atomic nucleus, while the biological and chemical reactions are the same. Substances not foreign to the body in the form of isotopes by the counting of radiation impulses can be demonstrated in such small quantities that no disturbance of the osmotic balance occurs.

In a first series of experiments an orthophosphate solution, produced in the pile at Harwell, with P^{32} as the label and a half-value

	NACH 37' TIER N°V	NACH 51' TIER N°II	NACH 58' TIER N°VI	NACH 77' TIER N°IV	NACH 90' TIER N°VII	NACH 175' TIER N°IX	NACH 250' TIER N°X	NACH 360' TIER N°XI
BLUT	1075,0 \pm 14,0	934,0 \pm 9,0	1010,0 \pm 15,5	1220,0 \pm 10,1	2490,0 \pm 25,0	2575,0 \pm 25,0	2750,0 \pm 23,0	3020,0 \pm 22,0
KA.WA.	493,0 \pm 7,8	440,0 \pm 3,7	414,0 \pm 4,6	744,0 \pm 6,0	1530,0 \pm 25,5	2170,0 \pm 28,0	2690,0 \pm 21,0	2840,0 \pm 32,0
LIQUOR	485,0 \pm 16,5	523,0 \pm 10,6	445,0 \pm 5,7	704,0 \pm 6,4	1155,0 \pm 9,5	2090,0 \pm 20,0	2990,0 \pm 18,0	3310,0 \pm 30,0
PERILYMPHE	104,0 \pm 3,6	87,0 \pm 3,5	117,5 \pm 3,2	232,0 \pm 6,1	406,0 \pm 6,4	1265,0 \pm 28,0	2042,0 \pm 30,0	2700,0 \pm 30,0
ENDOLYMPHE	24,0 \pm 4,5	46,5 \pm 3,1		55,0 \pm 4,4	80,0 \pm 5,0	195,0 \pm 11,1	206,0 \pm 12,0	308,0 \pm 15,8

Fig. 15.—The concentration of radioactive sodium in blood, aqueous humor, cerebrospinal fluid, perilymph and endolymph at various intervals following the subcutaneous injection of a sodium solution.

time of 14.3 days, was used for subcutaneous injections on guinea pigs (Fig. 14). These experiments showed strikingly low concentrations of phosphorus, not only in the perilymph but also in the aqueous humor and the cerebrospinal fluid as compared to the phosphorus level in the blood. In the blood the phosphorus rapidly combines with organic substances and a part of it is changed into the hydrated form, leaving for diffusion a considerably smaller inorganic rest. Furthermore, the phosphorus very quickly wanders from the intercellular spaces into the cells. Further experiments were, therefore, performed in this series with 1.5 to 3 ccm of a sodium chloride solution, containing Na^{24} as the label, with a half-value time of 14.8 hours. The activity of the quantity applied lies around 400-800 μ C. As in the experiments with phosphorus, samples of cerebrospinal fluid, aqueous humor, blood, perilymph and endolymph were taken at various times. The short half-value time necessitates correction of determined values. In order to obtain reliable results (Fig. 15) a hole was drilled into the scala vestibuli of the basal turn near the oval window. A second hole (Fig. 16A) was drilled into the scala tympani of the basal turn to avoid subnormal pressure. Once the perilymphatic spaces of the cochlea were emptied the round window was widened by lifting up a lid of bone at the end of the scala tympani, and the window membrane was removed. Thus a clear total view of the basilar membrane of the lowest turn was obtained (Fig. 16B). Then the whole area was dried with strips of filter paper,

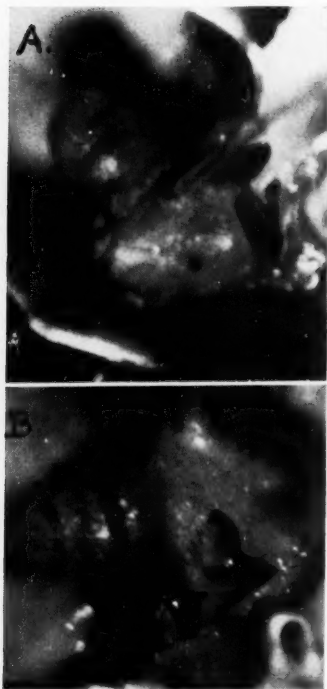


Fig. 16.—(A) A photograph of the holes bored into the basal turn of the cochlea. (B) View of the basilar membrane of the lower turn of the cochlea obtained after lifting up a lid of bone at the end of the scala tympani and removing the round window membrane.

and under the binocular microscope a very fine glass capillary was carefully brought into contact with the basilar membrane. Due to capillary 0.2 to 0.4 mgm of endolymph entered the glass tubule.

Fig. 15 indicates the concentrations of radioactive sodium found in these fluids at various times. Thirty seven minutes after subcutaneous injection sodium was found in all fluids. Initially the sodium level of the perilymph was considerably lower than that of the cerebrospinal fluid. On the other hand, the scala vestibuli shortly after injection, already showed a sodium level which was so high that the sodium could not have entered the scala vestibuli from the cerebro-

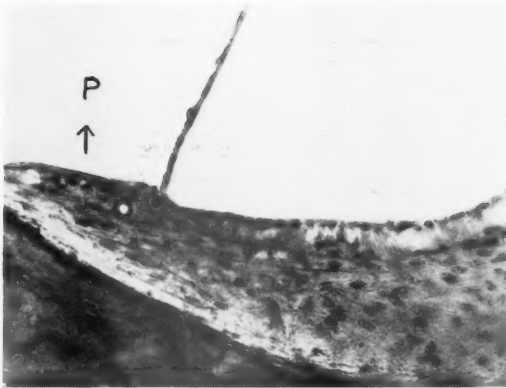


Fig. 17.—The site of origin of the perilymph in the guinea pig according to Mygind.

spinal fluid via the aqueduct in accordance with Fick's law of diffusion and model experiments. The perilymph, therefore, is not exclusively derived from the cerebrospinal fluid. There was initially a great difference between the sodium level in the perilymph on one hand, and in the cerebrospinal fluid and aqueous humor on the other. But this difference gradually became smaller until the sodium level of the blood was almost reached. This equalizing process leads us to believe that the secretion of sodium takes place principally in the same manner in each of the fluids, namely by ultrafiltration. The irregular initial differences, therefore, have to be related to the differences in size of the areas of exchange. The sites of ultrafiltration of the cerebrospinal fluid and the aqueous humor are known. According to Mygind (Fig. 17), the sites of origin of the perilymph of the guinea pig are the surface of the spiral ligament, extending beyond the stria vascularis into the scala vestibuli, and that part of the ligament which below the basilar membrane borders on the scala tympani. The sodium level of the endolymph is an altogether different matter. In all experiments the sodium level here was found to be merely a fraction of the level in the perilymph. This great difference could be found even six hours later. Despite the large number of blood vessels in the stria vascularis, ultrafiltration, therefore, does not seem to play any important part in the secretion of

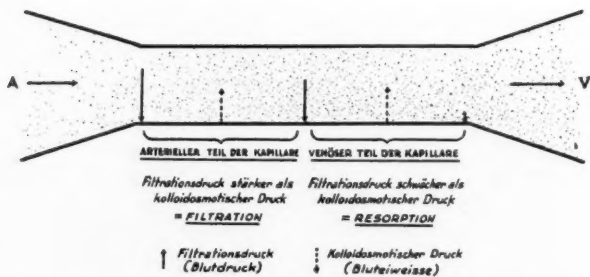


Fig. 18.—Schematic representation of the filtration and resorption of fluid in the region of a capillary according to Starling and Landis.

Production and Resorption of Endolymph during destruction of the organ of Corti

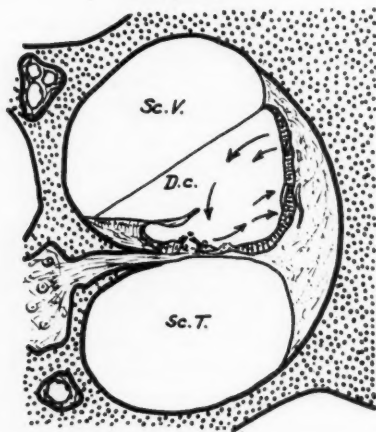


Fig. 19.—Production and resorption of endolymph during destruction of the organ of Corti according to Rüedi.



Fig. 20.—Particles of detritus from the disintegrating organ of Corti seen lying on the basilar membrane following overstimulation with a pure tone of 1600 c.p.s. at a sound pressure of 140 db over a period of 64 hours.

sodium into the endolymph. Here the surface epithelium appears to form a selective, protective barrier. The endolymph, therefore, is not produced through filtration, but through a specific secretion of substances necessary for the nutrition of the organ of Corti. To guarantee the safety of this very differentiated metabolic process, the secretory organ and the sensory organ supplied by it are enclosed in the fluid of the ductus cochlearis. It is true that the membrane of Reissner had been shown to be permeable for molecularly dissolved substances in diffusion experiments. But as long as the fluid exchange between the stria and the organ takes place with a certain speed, a certain amount of permeability of the membrane does not make any difference. Normally the metabolic products of the organ of Corti are at least partially resorbed in the sulcus spiralis externus. This would form a circulatory system over the shortest route, running across the direction of the turn.

In this connection we now shall take another look at the dilatations of the vessels which accompany the disintegration of the organ of Corti due to such different causes as overstimulation and intoxication. The regular localization of these alterations in the same area of the cochlea also corresponds to a circulatory system extending between the stria and the organ of Corti in a direction across

the course of the turn. Swelling of capillary loops of the corresponding segment of the stria vascularis appears to be the first response to the disintegration of any part of the organ of Corti. This opens the normally coherent surface epithelium at various points. Whenever the endothelia of the vessels have come into direct contact with the endolymph, ultrafiltration next to specific secretion should also be possible. According to Starling and Landis, ultrafiltration in any capillary system is based on blood pressure differences between the arterial and venous portion of the capillary on one hand, and the colloid osmotic pressure of the blood proteins on the other (Fig. 18). This leads to filtration in the first, and resorption in the second portion of the capillaries. In this manner the completely dissolved parts of the organ of Corti, rapidly disintegrating after trauma or intoxication, may be resorbed along the disrupted surface of the stria. This assumption is open to objections on the grounds that the pores of even dilated capillaries are still too narrow for protein molecules of a molecular weight of more than 1000, if the smallest vessels of the inner ear react similarly to the capillaries of the meninx and the eye. In the removal of large particles, therefore, macrophagic endothelia must play a part next to ultrafiltration.

In our large number of control experiments we could never find any signs of increased phagocytosis in the saccus and ductus endolymphaticus of these animals. Frequently, however, single detritus particles (Fig. 19) are found lying on the basilar membrane at the former site of the organ of Corti. From here the cell detritus can be traced laterally beyond the equally degenerated cells of the sulcus externus to the surface of the stria vascularis where minute particles (Fig. 20) appear partially anchored in the walls of the blood vessels.

This spatial relationship between cell detritus and stria capillaries also points to an endolymph circulation running across the direction of the turn.

Histological observations alone, however, no matter how clear they are, cannot give absolute proof that resorption and phagocytosis actually take place on the stria surface.

Secretion experiments with isotopes on guinea pigs injured by overstimulation or intoxication might be one of the ways to get this proof, but they take very long and cost a lot of money. Never-

theless, further investigations of the functions of the stria are not of theoretical interest only. They might help to explain the still unknown etiology of certain types of inner ear deafness, I am thinking particularly of Ménière's disease.

At this point I must end this incomplete account with suppositions and suggestions, well knowing that wishful thinking is the most dangerous luxury in scientific work.

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DISCUSSION

DR. LEROY M. POLVOGT: Mr. President and Members, and Guests; the findings that interested me the most were the lesions due to poisoning by streptomycin and quinine. I noticed the lesions were in the lower basal coil. If these poisons are conveyed by the blood vascular system through the stria why do they not affect the whole cochlea at once, not just the lower coil?

One easily understands how in a case of gunfire a particular area in the cochlea will be destroyed that corresponds to the pitch of the explosion, but in a poison transmitted by the blood vascular system, I would like to know why it does not affect the whole cochlea at once.

DR. MOSES H. LURIE: I wish to thank Dr. Ruedi for the beautiful presentation and for the considerable research because nobody knows better than I do the meticulous work and the care necessary in these experiments for they can easily go wrong.

I am particularly pleased that he confirms what I have found before, that the Organ of Corti disintegrates molecularly, just simply melts away in the scala media. We have never seen any phagocytosis or any indication of inflammatory reactions in the Organ of Corti.

In regard to the stria vascularis, we have also noticed a great many changes, but during the war when we were doing a good deal of this work, the stria was not studied because the Organ of Corti was our chief interest. Now we are beginning to restudy all the animals that we have to see if there is any real correlation. At the present time we cannot correlate the damage in the stria vascularis with the damages that occur in the Organ of Corti. In some cases the stria vascularis shows marked pathological changes. In other places it shows practically no changes.

I believe the reason we have changes in the Organ of Corti with toxic conditions in the first turn is because these animals are not kept in a soundproof room. As long as the animal hears, it is going to respond in its most sensitive part of the basilar membrane which is the first turn. Now being very sensitive and toxic, the Organ of Corti will degenerate quicker in this region than in any other portion.

We have observed in some of our animals that once the Organ of Corti is destroyed either by drugs or by trauma, that disintegration continues and if you keep the animal alive long enough, eventually all the Organ of Corti tends to disappear. Why the last remnant is up at the helicotrema is because that is the least sensitive part of the Organ of Corti.

The position of particles in the scala media, that Dr. Ruedi has described, I have seen some of them at the region of the stria vascularis. One must be very

careful in interpreting that because the way you fix your cochlea and the way you keep your cochlea after the fixation may make gravity play a part in the different positions of these particles in the scala. We notice that very often gravity will pull various structures one way or another after fixation.

I think this has been a most instructive presentation. I wish to thank Dr. Ruedi again.

DR. JEROME HILGER: When one confines his investigative or inquiring endeavors to clinical fields, he appreciates that his contributions are very inept compared with basic contributions as those here presented by Dr. Ruedi. This basic work answers many questions that one cannot clarify by clinical work alone.

There are several things that I would like to ask you, Dr. Ruedi:

When hair cells in the basal turn are damaged and thereupon the stria vascular area of the basilar turn responds by dilatation and epithelial disintegration or spreading, do you assume that there is possibly an axon reflex type of activity from the damaged neural cell area through the immediate neurovascular segment in that part of the turn? It is true in other parts of the body that axon reflexes play an important part in limiting vascular response to a particular region.

Another question: what in your opinion is the value of ionic restriction such as sodium restriction in the clinical approach to these problems? By your work it would appear that the secretory activity of the epithelial barrier when damaged is replaced by a simple diffusion principle.

Another question that I should like to ask: by your histological observations is it possible for the problem that we are familiar with as endolymphatic hypertension to be in some instances perilymphatic hypertension? Mygind has suggested this in his guinea pig studies. Does the human histology lend itself in some instances then to perilymphatic hypertension rather than endolymphatic hypertension? Because of this possibility we have been prone in discussing and presenting our cases to call it lymphatic hypertension, because of our own ineptness in being able to properly define and allocate it as endo- or perilymphatic.

We have also, Dr. Ruedi, one difficult clinical problem. We keep insisting from clinical observation that the most difficult inner ear case to treat is one that is hypertensive, because fluid which forms in the cochlear duct or in the perilymphatic space creates a situation of hypertension which makes resorption of it extremely difficult. We repeatedly see cases with the wide swinging type of hearing loss that we showed earlier in the week where we are defied in our efforts to get the lymphatic tension out of the inner ear. Is it your impression that the cluttering up of the resorptive capillaries by the detritus from Corti's organ is part of the reason for difficulty in resorption?

DR. BENJAMIN H. SHUSTER: I am sure you must be all as intrigued as I was by the discourse of Dr. Ruedi. He is one of the very few men that could deliver a discourse on complicated physics, chemistry and experimental work and make it sound like a novel. He has made things quite clear.

There is one point I want some information on. Aside from the interchange of fluids, Dr. Ruedi discussed the effect of sound trauma and chemicals on the organ of Corti, plus the changes that took place. What intrigued me most was the secondary changes that took place after hours or days following such trauma. Dr. Ruedi mentioned other causes and I was hoping that he might specifically mention the word "trauma." Instead he just said "other causes." Whether he

has done experimental work along that line or not I do not know. He certainly must have some ideas on it. What I have in mind is this: practically trauma comes up in our practice all the time and I have had an experience a year or two ago in a case of a patient who was hit on the head and the question arose in court as to whether that deafness is the type that has been caused by this accident and what compensation is involved in the case. I believe Dr. MacFarlan was involved with me in the same case and it was very striking that the patient lost a little bit of hearing following the trauma and month after month audiometric reading found a little more destruction of hearing. Was I justified in court in stating that as the result of this original trauma certain degenerative changes have taken place subsequently in stages and quite rapidly and that the trauma was the cause of this man's deafness and entitled to compensation? The point I wish to make—is physical trauma just as effective and does it produce the same delayed degenerative changes in the organ of Corti as does trauma by sound and intoxications?

DR. HENRY L. WILLIAMS: I have been greatly stimulated by this presentation of Dr. Rüedi's because, if I understand him correctly, he seems to have overturned certain fundamental ideas about the endolymph. I believe that his thesis is that the endolymph is produced not by filtration from the stria vascularis but by exudation from the secretory epithelium of the cochlear duct. This I think is the statement of outstanding importance which has been made in this paper. If it proves to be true it will certainly upset certain ideas in regard to the therapy of Meniere's disease which have been presented both by Dr. Hilger and myself.

I question somewhat if the evidence that he presents conclusively supports this position. You remember long ago that Dr. George Shambaugh described a series of glands in the sulcus externus, which he believed had a secretory function. I know that these glands have been neglected since 1907 but in talking over this problem of the physiology of the endolymph with Dr. Wakim at the Mayo Clinic, he stated that it is beginning to be generally accepted by all physiologists that secretions in body cavities are all a product of secretions of glands plus transudation from the capillaries.

In these particular experiments that Dr. Rüedi has undertaken is it not possible that the damage to the epithelium of the sulcus spiralis externalis which he has shown to be present interfered with these glands which secrete a watery saline solution which balances the transudated blood serum with its high protein content. Might not this be the factor which produces the changes in the endolymph which he has demonstrated. May it not be possible that in the normal individual the endolymphatic fluid may still be produced by conjunction of secretion and excretion by which rapid compensation for osmotic pressure changes may be secured as is the rule in other body cavities?

I know this is reasoning by analogy and I am on dangerous ground. Yet in physiology one does not see nature changing her physiologic mechanisms. One sees nature repeating functional patterns time after time in different areas and not changing to a different physiologic mechanism as different organs are developed.

PRESIDENT DAY: Dr. Hawkins, would you care to discuss this paper?

Dr. Hawkins!

DR. J. E. HAWKINS, JR.: Mr. Chairman, I have certainly very much enjoyed Dr. Rüedi's presentation, and I am very grateful for the opportunity to come here and hear it.

However, there are certain facts about streptomycin which I think are relatively little known and some of which I think must be taken into consideration when one discusses the effect of streptomycin and related substances on the ear

One of these points is, in our experiments we have had to distinguish rather carefully, at least our experiments have distinguished for us, between the effects of noise and quinine in particular on the ear and the effects of streptomycin, dihydrostreptomycin, hydroxystreptomycin, neomycin and alone, among other drugs, ascaridol. In electrophysiological studies of hearing—and I am talking particularly of experiments in the cat and guinea pig—we have found that the immediate effect of exposure to noise of high intensity is upon the action potentials of the auditory nerve and not upon the aural microphone effect of the cochlea. Noise selectively depresses the impulses passing up the auditory nerve and affects the microphonic only when much higher levels of noise are used. This refers to pure tones of course, whereas the effect of streptomycin, which we measure when we record the microphonic and action potentials on response of the cochlea from the round window, is specifically upon the microphonics and the first thing that we see is a depression of the maximum response of the cochlea to a pure tone, but at the same time the response of the nerve to clicks is almost fully preserved. Now that is a surprising and to us still rather an anomalous situation, which we don't know entirely how to explain. However, it suggests one of two things, either that there is a very large margin of safety in the amount of microphonic present and that necessary to stimulate the nerve, if one assumes that the microphonic does stimulate the nerve and/or it means that the microphonic has nothing whatsoever to do with the stimulation of the nerve.

Another point that I should like to make is that streptomycin, of course, affects the vestibular system primarily in all of the species which we have examined. We have had very little luck unfortunately in the guinea pig. The rodents are much more resistant to streptomycin than the cat, dog, monkey and apparently man also. Of the rodents we have examined—and we studied the mouse, rat, guinea pig and the rabbit—the guinea pig is the most resistant to streptomycin and I think Dr. Ruedi would probably find that if he used cats he would have considerable less difficulty in producing these changes with streptomycin than he must have with guinea pigs.

Another point, not so important nowadays, is the purity of the material used. It is, of course, highly important that the changes we observe not be attributable to extraneous substances in the streptomycin sample but rather to streptomycin itself. Therefore I think it is of the greatest importance that the streptomycin be of purity at least approaching 95 or 96 per cent.

I think also that Dr. Rüedi would find it possible to increase the efficiency of his studies of the circulation of the perilymph in particular if he resorted to intracisternal injection of material. I base that rather in the form of a question and ask if he has made such studies. We find, for example, with dihydrostreptomycin the effect of the material is enormously enhanced if one gives it intracisternally rather than subcutaneously or intramuscularly.

I should like to say also that these various antibiotics pick out different portions of the ear in a way which is still unaccountable. Whereas streptomycin affects the vestibular first and later the auditory, in our experience the dihydrostreptomycin affects the vestibular system at a later time and only very much later affects the auditory. We find that the dihydroxy, which differs in one hydroxyl group, has a greater selectivity for the organ of Corti. Neomycin picks

out the organ of Corti and leaves the vestibular system almost unaffected, and I think this is a very difficult thing to explain.

Finally, I should like to remind you of the fact that streptomycin is itself very poorly diffusible and it is excreted from the body, through the glomerulus of the kidney but it is not excreted so far as we know by the tubules of the kidney. So I wonder whether it is possible that it can be secreted by the stria vascularis. I am still inclined myself to think rather that it must come in very large part via the cerebrospinal fluid.

Thank you!

PRESIDENT DAY: Is there any further discussion? If not, I will ask Dr Ruedi to reply to these discussers.

PROF. DR. LUZIUS RÜEDI: Mr. President and Gentlemen, I thank you that you have been so kind in discussing this paper.

The first question of Dr. Polvogt has been answered by Dr. Lurie. The only explanation I can give you for the localized destruction of the organ of Corti in the first turn by streptomycin and quinine, and by over-stimulation of sound, is that at least at the end of the first turn this localized destruction might have a relation to the sensitivity of the organ in this region. I think everybody agrees with this idea that the lower part of the cochlea is the most sensitive part—Dr. Davis is here and he knows most about that—so if you have sound arriving in the ear during your experiments and this part is working and being very sensitive it may be hurt first. That is a theoretical explanation and I cannot prove it.

Dr. Hilger asked me how I can explain the relationship between the organ of Corti and the opposite stria vascularis, how these two parts correspond. I say I don't know that either, if it is a purely chemical humoral relation or in what way the nervous system is taking part. I don't know that.

Then Dr. Hilger asked me if we think that sodium is something which should not get into the inner ear and that is why it is retained by the stria. It seems to be and it is possible that if the stria does not function normally that instead of a very specific secretion taking place some filtration takes place and some material gets into the fluid of the endolymph which should not be there, and that may be sodium, and then this acts on the function of the organ of Corti. That is one of the problems which has not been studied but needs to be.

I believe that the chemical composition of the endolymph is extremely important for the normal function of the organ of Corti and that is why, if I might now jump to Dr. Williams' remarks, I do not think that you can compare the organ of Corti sitting in this endolymph having no blood supply by itself with any other part in your body. That is a very special arrangement and the nutrition of this organ, which has no blood vessels but which is supplied and lives as far as oxygen is concerned, as far as any other chemically needed stuff is concerned lives on the things which are brought to it, must come across the space within the endolymph. At least that is the idea we have about it. Maybe that is wrong too. But as long as you have that idea that the organ of Corti depends on the endolymph, then I feel that the endolymph must be equalized as far as the content is concerned up to the finest little changes, especially as far as oxygen is concerned. I feel all of us at one time or another have these little noises in

the ear, which come and then disappear again. I think those are just slight changes maybe in the oxygen supply going through the stria.

We never saw, Dr. Hilger, in our animals signs of a perilymphatic hypertension but that does not prove it does not exist. What we find now in some cases of Ménière's, this large dilatation of the saccus is just the last stage of a process which I think goes on in many cases only functionally without leaving any organic traces, so while it is possible that there may be hypertension in the perilymph, as you have proven hypertension in the endolymph, no evidence of it has ever been observed.

Dr. Shuster asked me a very interesting question. He asked me if the effect of head trauma may damage the inner ear too. Six years ago I had three extremely nice co-workers. One is my successor now in Bern, Dr. Esher. They were ready to go through an experiment, and with a hammer, I hit them hard on the mastoid process, and then they all had a very typical Cv drop of about 35 db, disappearing within 24 to 48 hours. That is the proof for the acoustic trauma following a head trauma or going on with a head trauma. The acoustic trauma in these cases I believe was just the loud noise produced by the blow going into the ear, doing the same as if a gun report had taken place before you. If you go farther along and hit the head harder, then you get the same thing in the inner ear as you may have if you have an explosion. One of your men, a co-worker of Dr. Lindsay, did beautiful work on that in experiments on cats, where he showed after trauma or very hard blows on the head there was blood within the scala tympani, scala vestibularis. As soon as you have blood in there then anything can go on. Then you may have a slowly starting destruction of the organ of Corti. You see tears in the organ of Corti, and you see them even in the stria vascularis. I saw in several men killed during the war by explosions of hand grenades, in such accidents, that behind an intact drum of the ear there was a tear of the basilar membrane in the inner ear. That is an explosion, where there is a very great pressure destruction, now without selection as to the sensitive parts of the ear. So, Dr. Shuster, I think in your case you were absolutely right to take it for granted that this man could have had trauma touching his ears.

I am practically through. Dr. Hawkins is, as you all know, one of the best men working with streptomycin, and I am very grateful that he told me to try several things. I am especially grateful for one thing he mentioned and that is the intracisternal injection of streptomycin. We did not try that. We were interested in the stria and we believe it comes through the stria. We believed it came there first but we may do it the other way around too. I know quinine first diminishes the nerve potential and only later the microphonics, whereas streptomycin first touches the microphonics, which speaks for beginning damage in the periphery. But it is possible that quinine not only damages the peripheral organ but also damages the ganglion cells. Quinine does many things besides damage in the ear and that could be an explanation why it always starts at the electrical potentials of the nerves.

I think that is all, and thank you once more.

SYMPOSIUM ON AUDIOLOGY

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LXXXIX

ADDRESS OF THE MODERATOR

S. RICHARD SILVERMAN, PH.D.

ST. LOUIS, MO.

Mr. Chairman, members of the Society and guests, in his Presidential Address of last year, which I read recently, Dr. Meltzer devoted one paragraph to the subject of audiology, and in it he said in essence we are going to have to watch this thing. This Symposium is an attempt to implement his recommendations through discussion.

It is well, I think, in setting the stage for the Symposium to examine certain factors which have led to an upsurge of interest in the field of hearing and deafness. Some of these factors were called to your attention this morning by Dr. Day, and they were there by implication in the other presentations, particularly in Dr. Kobrak's.

I should like to cite some specific factors that I think have contributed to this interest. I should like to point out here that this does not imply, as we well know, that nothing has happened prior to the coining of this term "audiology." I should say one major factor contributing to this interest has been the evolution of the fenestration operation. I speak of that influence not from the point of view of the surgeon and the evolution of the surgical technics involved. I am not competent to speak on that. But I feel that it has as a very valuable byproduct caused people, who heretofore had not been interested, to examine the physiology, the anatomy and the complete working of the auditory system. However one wishes to assess the value of the operation, I think it is undeniable that it has created a good deal of interest in the field of hearing. It has actually stimulated, I feel, some basic investigations in the field.

A second major factor is the development of finer measuring instruments. The electro-acoustic instrumentation available to us today enables us to make finer and finer measurements and enables us to quantify some of the concepts that we had to deal with previously in a qualitative manner. When we mention electro-acoustic instru-

mentation, we should mention the hearing aid and the influence that it has had.

A third major factor has been the development of new tools for examining the auditory system. Among these are the electrophysiological tools and then, as you saw this morning, cinematography. As we look back in the history of science, we always see, as new tools are developed, investigations become broadened and intensified. Improved technics have also made their contribution in the psycho-acoustic studies that have held such a prominent place certainly in auditory theory.

Another major factor, cited by Dr. Day this morning, was the wartime rehabilitation projects which brought together the peripheral workers, who are no longer peripheral, for a conjoint and integrated attack on the problems of deafness. You are familiar with that story and I need not elaborate it to this audience. But it seems to me that underlying all of these specific factors is the increasing social awareness of our people. This goes back to before 1933. If one examines the curve of social acceptance of responsibility for the handicapped, one sees that it is upward. One sees the increase in support, both from government and private sources, for interest in the handicapped and the hard of hearing have been included in the movement. I think that will relate to some of the audiological developments of the future.

Now let us turn our attention to audiology per se. You will notice that I will make no attempts to define it. I hope some kind of sensitivity to it will emerge from this meeting. As an emerging area of professional interest, audiology has had a varied orientation, depending upon the context in which it is practiced. Organization-wise we see it either as an appendage to, or thoroughly integrated with, the university department of speech, as a non-medical adjunct to the department of otolaryngology, as an academic area drawing upon the traditionally constituted department, as a self-sufficient unit functioning independently, or in various other forms too numerous to mention, and I think this is critical for this group. Personnel-wise the pattern is similarly variegated. Speech correctionists, teachers of the deaf, otologists, acoustic physicists, clinical and experimental psychologists, physiologists, public health experts and others grace the audiological scene. This is a wholesome situation for a developing specialty since it mitigates against premature delimitation of its scope and it furnishes us with the useful empirical tests of the effec-

tiveness of certain types of organizations and skills in coping with the problems of the hearing handicapped.

On the other hand, we must take heed lest our enthusiasm lead us to audiological amorphism through which the specialty comes to mean all things to all men and we are forced to say, "You pays your taxes and you takes your choice."

I feel that the proper scope of the specialty is of more than passing concern to those of us who must organize, administer and seek support for programs in audiology. Aside from institutional and formal programs, I think perhaps the most critical question is the relationship of the practicing otologist to what is still an amorphous concept.

This afternoon we want to set the stage with a series of brief addresses on subjects of audiological interest, but we feel that the success of this meeting will depend upon you, the audience. We hope to get the corridor gossip up to the stage and get your opinions and your comments on these problems.

Our first presentation this afternoon will be "Minimal Requirements of Equipment for Audiological Work," by Dr. Scott Reger of the State University of Iowa.

CENTRAL INSTITUTE FOR THE DEAF
AND
WASHINGTON UNIVERSITY MEDICAL SCHOOL.

MINIMAL REQUIREMENTS OF EQUIPMENT FOR
AUDIOLOGICAL WORK

SCOTT N. REGER, PH.D. (By Invitation)

IOWA CITY, IOWA

Before I begin my talk, I want to say that I hope I am able to avoid the impression that I, an individual without medical training, am dictating to the otologist in regard to the equipment he must possess for audiological work. I propose merely to mention the potentialities of a few pieces of equipment which have been demonstrated by many individuals to possess positive value in clinical auditory measurement problems. Incidentally, I hope that the phrase "audiological work" has not generated confusion. In this paper "audiological work" means careful and thorough testing of the hearing function.

Before becoming too immersed in equipment, the proposition that instrumentation is a means to an end and not an end in itself should be considered. Unless the operation and limitations of the equipment are known and observed, more and bigger mistakes will be made with equipment than without it. It is preferable to use simple equipment with clinically useful accuracy than to attempt to employ complex procedures with accompanying uncertainty about the results. An individual who has learned how to administer and interpret tuning fork tests from clinical experience, but who knows little about audiometry is a much safer man with forks than with an audiometer. Dr. Lempert made a profound statement about a year ago when he stated that the otologist has less need for new functional hearing tests than for a better understanding of the tests he already has. Additional tests, of course, are justified only when they contribute useful information not supplied by already existing tests, or provide the same category of information with a desirable higher degree of accuracy.

From Department of Otolaryngology, University Hospital, Iowa City, Iowa.

With the calculated risk of displaying no originality in the designation of equipment which has been found useful by many individuals and clinics for audiological work, my list is headed by a speech audiometer. This instrument enables the examiner to determine the patient's hearing loss for speech in decibels in relation to either the "normal" speech reception threshold intensity level or any other designated zero reference level. By plotting the number of PB words perceived at different intensity levels a speech function curve can be obtained which indicates the patient's ability to perceive speech at above threshold levels. The position and slope of this curve in relation to the normal, reveal data of diagnostic significance which, of course, are to be interpreted in relation to other hearing tests and to the patient's ear, nose and throat history and physical examination. The slope of the speech function curve may be significant in relation to recruitment and also provides data for a determination of the SAI, which is an average numerical evaluation of the patient's ability to perceive low, conversational and high level speech. In my opinion the SAI provides a much more accurate and realistic evaluation of hearing disability than any other method in use at the present time and eventually will replace other methods of determining compensation for auditory disability.

A speech audiometer, when equipped for free field testing, is indispensable for use in the selection of hearing aids and for checking hearing aid performance. The speech audiometer also forms the basis of a well known test for psychogenic deafness which probably will be described by Dr. Doerfler, one of the co-inventors of the test, later in the program.

Very often a clinically useful test of auditory acuity can be obtained on children with a speech audiometer on whom threshold measurements cannot be obtained with a pure tone audiometer. Incidentally, a speech audiometer equipped for a free field testing is the best device I know for demonstrating to a parent the presence of a significant impairment in a child if the parent doubts the existence of the loss. People often have to be shown, and the speech audiometer is unexcelled for the purpose. Similarly, a speech audiometer can also be used to demonstrate to reluctant parents or relatives the difference in speech intensity levels that can be perceived by a patient with and without the use of a hearing aid.

If a speech audiometer is equipped with a good bone conduction vibrator, the instrument can be used for the measurement of speech by bone conducted sound.

The above assortment of tricks by no means exhausts the capabilities of the speech audiometer but does indicate its potentialities.

Many pure tone audiometers already in use can be modified to lead a double life by equipping them with accessories which permit the reproduction of speech in the audiometer earphone. An additional power amplifier and loud speaker are needed for free field testing. If a microphone for live voice testing is desired, two separate testing rooms and an additional talk-back amplifier are necessary. The testing can be done in a single room if the speech is amplified from phonograph records. The usual masking precautions must be observed while making measurements of speech reception thresholds and obtaining speech function curves.

Speech amplifiers can now be obtained from a few commercial sources and suitable phonograph records also are available. Fortunately the American Standards Association is compiling Standard Specifications for Speech Audiometers at the present time. The publication of this report will constitute a significant milestone in the advancement of speech audiometry.

The second instrument on the list is the pure tone audiometer, in spite of the fact that my discussion of the speech audiometer has left very little for the pure tone instrument to do. The pure tone audiometer reveals data about the acuity at frequency levels above the speech frequency range. Pure tones are most convenient for detecting the presence or absence of diplacusis. Bone conduction measurements can be made at higher frequency levels than are possible with forks or with the speech audiometer. The pure tone audiometer can be used for the measurement of recruitment and can be modified or provided with an attachment for administering a foolproof version of the Stenger test for the detection of unilateral malingering. Relationships between test results obtained with the speech and the pure tone instruments often are needed to differentiate between so-called central and peripheral auditory dysfunction. A complete study of the hearing process is obtained most readily at the present time by the combined use of both types of audiometers.

At the present time the pure tone audiogram also serves as an indication of disability.

At the risk of losing my union card in the Audiology Association, which seemingly has absorbed me also, I throw discretion to the winds and boldly place some tuning forks among the list of equipment for audiological work. The individual who knows the technic and interpretation of the fork tests, but who does not trust an audiometer because he does not understand it, can become familiar with the audiometer most readily by observing the similarities between the results obtained with the two types of instruments. When he learns that the results are comparable, and possibly identical, he is well on his way to understanding and appreciating his electronic gadget. In spite of technical advances, the tuning fork remains the simplest and most foolproof vibrator for the measurement of bone conduction acuity. Until a sturdy audiometer bone conduction vibrator is developed which maintains its calibration over long periods of time, I cannot criticize adversely any person who insists on making his bone conduction measurements with forks.

Finally, a few noise makers or percussion toys should be available with which to observe the reactions of young children to low, medium and high level sounds or noise.

Obviously no audiological research equipment has been included in the above list. The equipment requirements from the hobby or research standpoint will be limited only by lack of imagination or lack of funds.

The training and scientific integrity of the individual who uses the equipment is of much greater significance than the physical equipment itself. As indicated earlier, the use of precision equipment by an unprecise mind can lead to all kinds of astounding and confusing results, not to mention startling so-called research. It is impossible to take maximum advantage of the improvements in instrumentation unless the user is willing to spend time studying the physical principles, operation, limitations and interpretations of the test results. It may hurt a bit at first, but will turn into a pleasure as the feeling of frustration vanishes.

UNIVERSITY HOSPITAL.

XCI

HEARING AIDS: HOW THEY WORK AND FOR WHOM

IRA J. HIRSH, PH.D. (By Invitation)

CAMBRIDGE, MASS.

A hearing aid, strictly speaking, does not aid hearing—it only aids, or enhances, sounds. By a process of *amplification* the hearing aid operates on certain physical properties of sound; specifically, it produces a sound intensity in the ear canal that is greater than the sound intensity that first impinged on its microphone. The patient's hearing or hearing loss remains the same; it must—the hearing aid was not designed to change the pathology but only to change the sound so as to compensate for the hearing loss. Let me review briefly some of the principles of amplification and then discuss certain clinical problems in relation to these principles.

AMPLIFICATION AND ATTENUATION

Whenever a device makes a physical change appear greater at the output than at the input, the device may be called an amplifier. Thus amplification is exemplified, not only by the vacuum-tube hearing aid, but also by the public-address system, the audiometer, the speaking tube, etc. There can, of course, be as many different kinds of amplification as there are different forms of energy to be amplified.

Before we had vacuum tubes, sounds had to be amplified in a purely mechanical way, either by resonators or by devices like the speaking tube or the cupped hand behind the ear. The vacuum tube, however, provided a much more convenient and controllable method of amplification, but, of course, the vacuum tube has to do with the amplification of electrical energy only. Briefly, a small amount of energy change is supplied to the input or grid of an amplifying vacuum tube. The small changes in the grid voltage control the main current flow through the vacuum tube so that changes in the output voltage appear very large. Of course, we do not get something for nothing in this system, because the additional energy is

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supplied in a constant or d-c form by the batteries or other power supply.

Electrical amplification can be utilized for sound if we can convert the weak sound into electrical energy and then reconvert the amplified electrical energy to sound. This conversion or transduction is accomplished by microphones or phonograph pickups (in going from sound or mechanical energy to electricity) and by loudspeakers or earphones (in going from electricity to sound).

With these basic notions in mind, let us describe the components of a small wearable hearing aid.¹ A microphone, containing a diaphragm and a crystal, is activated by sound waves. These sound waves are converted into displacements of the diaphragm, which, in turn, applies alternating mechanical forces to the surfaces of the crystal. The crystal is a substance that develops electrical charges across its surfaces when a mechanical deformation is applied. The electrical output of this crystal microphone is applied to the grid of the first of several stages of vacuum-tube amplification. The final amplified electrical energy is fed to an earphone, which converts, usually on an electromagnetic principle, this electrical energy into a stronger sound than the one that originally moved the microphone's diaphragm.

Attenuation is just the opposite of amplification. In an electric circuit, attenuation is provided by energy-absorbing resistors. In mechanical circuits, attenuation is provided by frictional components. If a sound is first amplified by a certain amount and is then attenuated by the same amount, we have the same sound in the end. Both of these processes have to do with only one physical property of sound, namely intensity. You remember that the sinusoidal sound, or pure tone, that is produced by a vibrating tuning fork has two important properties: intensity and frequency. When such a vibrating tuning fork is placed in front of a hearing aid, the output sound from the hearing aid should have the same frequency as the input, but the intensity will be greater. If the same tuning fork is placed near a sound-absorbing wall and if we measure the sound on the other side of the wall, the frequency should still be the same but the intensity will now be less. These are examples of amplification and attenuation, respectively, of a pure tone.

The sounds of speech, which are of greater practical importance than pure tones, are more complicated in that they contain many

frequencies simultaneously. When we speak of amplifying the sound of speech we must introduce the frequency-vs-response characteristic of the amplifier in order to know whether or not our device amplifies all frequencies equally well. If it does, we say that its frequency-response characteristic is flat or that it is a 'high fidelity' amplifier. If, on the other hand, our amplifier favors certain frequencies over others, that is, it amplifies certain frequencies more than others, its response is 'peaked' in a certain frequency region and it has introduced frequency distortion. Another type of distortion that may be present is amplitude distortion but we shall disregard this type because it has been shown² that it has little effect on the intelligibility of speech.

A word about the decibel (db). The numbers involved in specifying amounts of amplification may become unwieldy. For example, the intensity of a sound from the earphone of a hearing aid may be 100, 1000, or even 10,000 times as great as the intensity of the sound that impinged on the microphone. To save writing all these zeros, engineers use the logarithm of the ratio of output to input intensities to specify the amplification. Specifically, the decibel is ten times the logarithm to the base ten of this ratio. Thus the three intensity ratios mentioned above would be 20, 30, or 40 db respectively. Note that these are the same units as we use to measure a hearing loss and there is a unique relation between the two kinds of measurement.

AUDIOMETRY AND HEARING AIDS

When and how do we say that a patient has a hearing loss? With an audiometer, we present to a patient a particular pure tone at a particular intensity marked 0-db Hearing Loss, which most people with normal hearing can hear a certain percentage of the time. Our patient, however, gives no indication that he is hearing the tone and so we increase the intensity 10, 20, 30, or 40 db until he signifies that he hears it. We note that at this frequency a given amount of amplification (i.e., the Hearing Loss) will permit this patient to hear as normal. When we make an audiogram, we usually determine the amount of amplification needed at each of about seven frequencies. It would seem, at first glance, that a hearing aid could be prescribed on the basis of this audiometric evidence. Simply design a hearing aid that will provide as much amplification as is indicated by the amount of Hearing Loss at each frequency. Would that it were so

simple as this "mirror-image" technique suggests! We shall see presently that it is not.

Speech audiometry yields a similar picture—the Hearing Loss for Speech is simply another way of saying the amount of amplification needed for a specified amount of speech intelligibility. This needs further clarification. When you stand 20 feet away from a listener and speak in your 'standard' voice, you assume that the intensity of the speech at the listener's ear is just about sufficient for him to understand your speech if he has normal hearing. If he does not respond, you increase the intensity of the speech at his ear by moving closer. In particular, whenever you halve the distance between yourself and the listener, being sure to hold the intensity of your voice constant, the effective intensity at his ear increases four-fold. The use of earphones and an amplifier that is fed by either a phonograph or a microphone is simply a refinement of this basic technique. In this latter case we can know the number of decibels that the normal speech intensity must be increased in order that the patient hear a given percentage of the speech. We simply note how much amplification has been introduced into the circuit. Again these audiometric results tell us the amount of gain that is required in order that the listener hear as if he had a normal threshold. We are always concerned with making sounds more intense than they are naturally in order to compensate for an attenuation in the auditory system. This last sentence brings us to the basic problem in the use of hearing aids today.

HEARING AIDS—FOR WHOM?

A great many hearing losses can be regarded as simple attenuations. When an auditory pathology involves attenuation, as it does in a blocked ear canal, otitis media, or otosclerosis, the prognosis for a hearing aid is good. The pathology results in a constant diminution, by a fixed number of decibels, of all entering sounds. To compensate for such an attenuative hearing loss we have only to boost the intensity of sound entering the ear canal by the amount by which it will later be attenuated in the pathological structure. With only minor qualifications we can say that a hearing loss that behaves as an attenuator—usually a conductive hearing loss—will be easily compensated by a hearing aid.

But there are many hearing losses that are not of the attenuative type—usually, but not always, nerve-type hearing losses. Of

course, the otologist has many ways of distinguishing between a conductive and a perceptive hearing loss, but in making a prognosis about a hearing aid we are not so much interested in the pathological diagnosis as we are in the way the auditory system responds to amplification (i.e., attenuative or non-attenuative). How do we decide that a hearing loss is not of the attenuative type? So long as we restrict our measurements to the threshold audiogram or even to a threshold of intelligibility for speech, we cannot answer this question. We must observe how the auditory system behaves as we increase the intensity of sounds above their threshold values. In clinical work, there are two kinds of supraliminal measurement that have been used, but more are coming to be used every day.

First, there is the relation between the psychological attribute of loudness and the physical attribute of intensity. Deviations from a normal relation between these two attributes are usually subsumed under the term recruitment.³ This topic could well take our entire time and we must pass it by because its relation to the intelligibility of speech remains so vague.⁴ Suffice it to say that since recruitment involves an abnormally rapid growth of loudness at those frequencies for which certain patients have hearing loss, it predicts very serious consequences for loudness of high-frequency tones that have been amplified selectively through a hearing aid whose frequency-response characteristic has been 'fitted' as a mirror-image of the audiogram.

Controlled speech audiometry has yielded a second practical way to distinguish between hearing losses of the attenuative type and hearing losses that are more complicated. And the recent formulation of a Social Adequacy Index⁵ has served to crystallize the basic notions involved. Briefly, we must recognize two independent properties of the auditory system in recognizing the sounds of speech. One has to do with sensitivity or need for amplification and the other has to do with the ability to discriminate among the sounds of speech when the intensity is sufficiently high. Clinically, these two properties are measured by using two different types of speech material and measuring two different response criteria.

Hearing loss for speech describes only the first of these two properties. It is the difference (in db) between the normal threshold of intelligibility and the patient's threshold of intelligibility. This threshold is defined as the intensity of sound at which a listener repeats correctly 50 per cent of a list of words that are presented to

him. The kind of speech that is used in lists for this measurement usually consists of relatively easy material such as sentences or two-syllable words.⁶ And as a matter of experimental fact, this threshold of intelligibility can be predicted very reliably from the average hearing loss at frequencies 500, 1000, and 2000 cps.^{7, 8}

The second property, the ability to discriminate the sounds of speech, is usually measured by presenting fairly difficult words at a relatively high intensity, an intensity so high that a further increase in intensity will not improve the discrimination. The Psycho-Acoustic Laboratory's phonetically balanced (PB) lists have been found extremely useful for this measurement.⁹ We measure the highest percentage of the words on a given list that a patient can repeat correctly at this high intensity. Persons with normal hearing as well as those with conductive hearing loss can usually repeat between 80 and 90 per cent of the words on such a list at an Intensity Level of 100 or 110 db. The non-attenuative hearing loss, however, is evidenced by a maximum score of much less than these amounts. It is clear from the nature of the testing procedure that amplification alone is not sufficient to improve this ability to discriminate and this fact clearly limits the usefulness of a hearing aid for such patients as far as the intelligibility of speech is concerned. When someone builds a hearing aid whose function it is not to amplify sound but rather to make the sounds of speech more discriminable, then we shall begin to recommend hearing aids for such patients with more enthusiasm. So far we have not enough information on the basic process of perceiving speech to guess at what such a device might be like. The only way that we have at present to increase the discrimination among sounds at a given intensity is a form of educational therapy known as auditory training.

SUMMARY

It is hardly news to this audience that, in general, persons with conductive deafness can get more help from a hearing aid than can a person with perceptive deafness. It would be presumptuous to take your time for that observation alone. Rather we have attempted to show why this observation is to be expected when we consider the way in which the hearing aid operates on sound in relation to types of hearing loss that we encounter. An important observation that follows from this discussion is that the very process of measuring the

hearing loss for speech and the maximum articulation score is actually a test made with a good hearing aid of controllable gain. In spite of the fact that wearable hearing aids have not the fidelity of most test equipment, it is clear that speech audiometry is a necessary step in the prediction of success to be enjoyed from a hearing aid.

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XCII

THE DEAF AND HARD OF HEARING CHILD

CLARENCE O'CONNOR, PH.D. (By Invitation)

NEW YORK CITY, N. Y.

The hundreds of children who are either born deaf each year or who become deaf very early in life must travel a long and frequently very lonely road in order to reach that goal we all seek, namely happy and useful citizenship. To them the philosophy that all men are born with equal opportunity is a cruel joke, for they must achieve their destiny against tremendous odds. Against a backdrop of silence, with ears closed to the priceless boon of spoken language, they must learn the art of communication, for this is the open sesame to successful achievement in our world. Through mediums less flexible than the miracle of hearing they must acquire the skills that they need in order to compete with their hearing brothers and sisters. And all of this they must do in a relatively unsympathetic world, for deafness, the unseen handicap, is an irritating handicap; somehow or other the hearing public cannot seem to comprehend the magnitude of the problems it presents.

A little blind child, groping his way through this rough world, inspires the sympathy of all, and rightly so. But the blind child becomes a relatively normal child because he can communicate normally and freely. We are proud of his achievements and also very tolerant of his shortcomings. Similarly our hearts go out to the crippled child as he moves haltingly through life, and when he learns to walk, even though defectively, we are proud and amazed at the small miracle that has been wrought. We are not impatient with him because he has not learned to walk as we do. In our daily contacts he, too, becomes a relatively normal child for he too can communicate normally and freely. How different it is for the deaf child. At the end of his long and tedious journey he too meets the hearing world with the ability to communicate with his fellow man. He has learned to speak but his speech, of course, is far from nor-

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mal. He has acquired the amazing skill of speech reading, but, of course, this skill is not as reliable for interpreting spoken language as is normal hearing. With him, too, a small miracle has occurred, but the miracle is lost in the wave of irritation that generally is induced in the unsympathetic listener because of the communication shortcomings that still exist. Because he has not learned to talk as we do we repudiate his achievement, reject him, and castigate his teachers for not having worked a larger miracle. Is this picture overdrawn? Not at all. It happens every day.

Several hundred years ago the deaf were considered uneducable. They had no legal status, were generally regarded as mentally retarded and were usually hidden away from the public by devoted, but confused parents. The earliest attempt to educate the deaf in America came with the establishment of a school at West Hartford, Connecticut in 1817. It was believed that this school would be able to meet the educational needs of all deaf children in the United States indefinitely. Soon, however, schools were established in other states to meet growing needs until, by the turn of the present century, nearly every state in the Union had at least one school for the deaf. These early schools were nearly all resident schools. As transportation facilities improved more and more, day schools or day classes were established to meet the increasing desire of parents to educate their deaf children in programs that would permit them to live at home. Today, therefore, there are 276 schools or classes in which 20,946 deaf children between the ages of three and 21 are enrolled.¹ Seventy-three of these are resident schools with an enrollment of 13,636; 158 are day schools or day classes with an enrollment of 5,609; and 45 are private or denominational schools with an enrollment of 1,701.

Prior to 1867 very little effort was made to teach the deaf to speak and read the lips. In that year two schools were founded that were destined to change radically the course of the education of the deaf in the United States. These were the Lexington School in New York and the Clarke School in Northampton. The philosophy advanced by these two schools was that all deaf children should be given an opportunity to learn to speak effectively and read lips. This was the beginning of oral education in this country. In the intervening years this philosophy has been gradually accepted by all schools for the deaf. Today, therefore, all deaf children have the

opportunity at least to begin their educational careers in an oral environment. In some schools, however, some of these youngsters are not given the opportunity to move through their entire program under oral instruction, but are given a large part of their education manually after they reach the age of ten or thereabouts.

A number of advances have been made in the past generation that have greatly improved the educational opportunities for the deaf. These progressive moves originated in and have consistently received their major impetus from the resident schools. Some of these advances are the establishment of nursery school programs for the deaf, improved techniques of developing communication skills and the utilization of residual hearing.

The first five years of a child's life represent the most prolific years of learning. Many schools for the deaf now recognize that this is true for deaf as well as for the normally hearing children and admit deaf pupils as young as three, and in some cases even younger. Youngsters who can be enrolled in these nursery schools may profit in at least two directions. Because of early contact with other deaf youngsters many personality complications that might develop if they were to remain at home in semi-isolation may be avoided. In addition, as a result of earlier instruction many have been able to reduce materially the three or four year educational retardation period that normally one may expect to find in the deaf.

In many schools a radical change has also occurred in the manner in which communication skills are developed. Speech, speech-reading and language are being presented in such a way as to develop early in the deaf child an interest in the communication values of language as contrasted with the traditionally formal approach which placed great emphasis on the mechanics of speech. This has been profoundly influenced also by the extensive use of hearing aids in schools for the deaf. It is well recognized now by most educators of the deaf that there are very few so called completely deaf children. Most have some residual hearing, and even though the loss may be severe or even profound some benefits will accrue to the child through its use. The major benefit will be in the direction of voice and speech improvement. Some interpretive benefits may also be expected for those children who may be called educationally deaf or severely hard-of-hearing with losses ranging from 60 db to 90 db. These youngsters can combine effectively their lip-reading skill with the use of

their residual hearing to develop a much improved ability to understand spoken language. This auditory training program which is given to all pupils in many schools for the deaf regardless of hearing loss is, of course, not designed to convert deaf children into hard of hearing children since by far the majority of pupils in schools for the deaf have losses in excess of 90 db.

DISCUSSION

Before I conclude this paper I should like to present a few questions, observations and suggestions, the answers to which or the discussion of which may lead us mutually to a better understanding of the problems that surround the education of the deaf. Some of these questions are:

1. "Can the deaf really learn to speak?" The answer is "yes." Not all will learn to speak equally well. About 30% may speak so well that anyone can understand them; about 40% will learn to speak effectively enough to be understood by most people; the other 30% will learn to speak to some degree but their speech will not be easily understood by the public. A visit to your nearest school for the deaf will give you an opportunity to prove to yourself that a large proportion of the deaf can learn to speak effectively.

2. "Do the above results justify the time and energy spent on oral education?" Definitely. Even those who speak most defectively can be understood by their relatives at least and in most cases by the people with whom they work. The real justification for this program, however, is found in the upper 30% who, though severely deaf, are able to move freely among their hearing fellow citizens handling the English language almost as easily as their hearing brothers and sisters.

3. "Is it not better for the deaf to attend day schools rather than resident schools?" All educators of the deaf agree that if a deaf child can attend a good day school for the deaf and thus live at home while going to school he should do so. The critical point in this question, however, is that the program be effective regardless of whether it is a day school or a resident school. In rural communities day schools for the deaf generally cannot be maintained. Single day classes likewise do not always provide the most effective program since variations in age, hearing loss and ability recreate the undesirable conditions of the little red school house. The most effective

program for the vast majority of the deaf is either a resident school or a day school with a large enough enrollment to allow for good grading according to ability, age and hearing loss. This type of program is generally better able to maintain a staff of well trained teachers, and through the combined progressive thinking of a large group of specialists can provide a better rounded program than can be found in isolated classes located in the regular school program.

4. "The deaf should not be confused with the hard of hearing." In general the presence or absence of relatively normal language will roughly classify children as either hard of hearing or deaf. Accompanying this type of classification, then, will be recommended educational programs designed to meet most effectively their respective needs. The educational program of the hard of hearing should be provided in or close to the regular school program for the normally hearing. The program for the deaf will be most effectively implemented in special schools or classes for the deaf wherein small classes and an abundance of individual help may be provided. Exceptions to this general procedure will be observed regularly. Certain borderline severely hard-of-hearing cases may need the benefit of the slower paced deaf program for a time at least and certain exceptional deaf pupils may similarly be able to meet successfully the challenge of the normally hearing program after they have established their language skills.

Through misinformation and through lack of experience with the complex problems of educating the deaf, that is those with severe loss of hearing, many people in audiological and hospital clinics, University departments of special education, and city and state directors of special education who have recently become concerned with problems of hearing loss, confuse the deaf with the hard-of-hearing. Recommendations are frequently made which imply that the deaf can all move through the regular classes for the normally hearing. The hard-of-hearing can do this. The deaf generally cannot. Such generalized recommendations have brought grief to many a deaf child and his parents. A thorough familiarity with the problems of both groups would make their guidance more expert and effective.

5. "There should be a better understanding of the value and the limitations of hearing aids." Too many doctors, clinicians and

salesmen are inclined to prescribe hearing aids for any and all children regardless of loss, and leave the impression with parents that the child's problems will all dissolve soon if they conscientiously wear the aids long enough. A hearing aid can never convert a severely deaf child into one who can use his hearing as a major medium of language interpretation. It can help his speech but it won't make him hear.

6. "There should be a better understanding on the part of all regarding the extent to which children with hearing losses can be integrated into the regular programs for the normally hearing."

In general most hard-of-hearing children, particularly those with losses of less than 60 db, can move along satisfactorily in or close to the program of the normally hearing child if he is given all the differential benefits he needs such as favorable seating, lip-reading instruction, a hearing aid and individual assistance. Conversely, in general, the deaf cannot do this and in most cases should not be forced into his situation. In an attempt to remove the deaf from conditions of so called isolation by placing them in the regular program, they are forced into a condition of much greater isolation, for they usually find that they are very much by themselves in a crowd; both lonely and frustrated. Incalculable educational and emotional harm may be wrought by sweeping generalizations of this nature.

The deaf child has found through bitter experience that his biggest problem in life is not his deafness alone. Frequently the unsympathetic, impatient and intolerant normally hearing public represent the source of his greatest woes. They cannot seem to sense the herculean task he faces in his long and arduous search for social and educational competence. They cannot seem to evaluate fully the scope of the miracle of a deaf child learning to speak without benefit of hearing. Will you through deeper understanding join with the educators of the deaf in their efforts to provide for this handicapped group the rich opportunity that is the inherent right of every child?

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XCIII

PSYCHOGENIC DEAFNESS AND ITS DETECTION

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PITTSBURGH, PA.

We hear only what is meaningful to us. The very fact that we exist as human beings brings about a delimitation of the possible stimuli of which we are conscious. We filter out of the world of noise in which we live only the particular sounds to which we tune ourselves, perhaps only those which have value in terms of survival. Certainly such a sifting is vital if we are to live in this world of billions of sounds.

Hearing is thus both a passive and an active process, and individuals who have difficulty in hearing without a comparable organic basis for this disability sometimes present themselves. This condition, psychogenic deafness, may exist as the basic cause for the hearing difficulty or as an exaggeration of an organic hearing loss.

The literature of the past century contains frequent references to cases of psychogenic deafness, psychological deafness, psychic deafness, pseudo-deaf muteness, neurotic deafness, hysterical deafness, central deafness, functional deafness and the like. For the most part these terms have been used synonymously and interchangeably. And almost invariably, the psychogenic deafness is described as being accompanied by associated disorders such as pain and cutaneous hypersensitivity, hemianesthesia, blindness, loss of smell and taste, vertigo, or amblyopia. These factors are described repeatedly as accompanying psychogenic deafness, and as a natural consequence, soon were looked upon as symptoms indicating the presence of psychogenic deafness, with a resultant shifting of emphasis in diagnosis from the hearing loss to a supposedly invariably associated concomitant.

It has only been since World War I that this classic picture has been questioned. Publications during the last ten years make little

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or no mention that sensory disorders are associated with psychogenic deafness.

The relatively large numbers of psychogenically deaf patients reported by the military aural rehabilitation centers during the last war raised certain obvious questions concerning the incidence of psychogenic deafness in the civilian population. Specifically, these questions might well be asked:

How frequently may an otologist in private practice expect to be confronted with psychogenically deaf patients?

What measures offer the best hope at present to identify these patients?

Data on the actual incidence of psychogenic deafness is available only from military sources. The figures given range from 10-20% of a series of routine admissions to military hearing centers. While it is true that some of these men were combat casualties, a large proportion were not exposed to any military stress other than the trauma experienced in wearing a uniform. It might be said that they were essentially civilians dressed in uniforms.

The University of Pittsburgh Department of Audiology has recently completed a survey of 30 leading audiology centers in an attempt to obtain up-to-date information on the observed incidence of psychogenic deafness in their routine patient load. It should be borne in mind that this patient load is a highly screened one, in which the majority of cases have been referred by otologists to the audiology centers, and therefore may not represent the true incidence at the level of the otologist.

In brief, the data in the case of adults showed that 53% of the centers reported encountering either no psychogenically deaf patients or merely an occasional one during 1950. Thirty-seven per cent reported an incidence of from one to five per cent, and ten per cent reported figures in excess of five per cent. The data on children showed 74% of the centers reporting few or no psychogenically deaf children, 21% reported an incidence of from one to five per cent, and seven per cent over five per cent.

These figures lend themselves to various interpretations, but they do indicate that while a majority of centers is uncovering few psychogenically deaf individuals in their routine case load, more than

one out of every three centers is finding routinely a sizable proportion of these cases. During the past two years our department has been able to demonstrate the existence of psychogenic deafness in 3% of our case load. We feel that a major cause of this discrepancy lies in the varying degrees of awareness of the problem on the part of otologists and audiologists, and the lack of routine utilization of several simple special techniques.

In our survey of hearing centers we found that a majority of those reporting few or no cases of psychogenic hearing loss utilized these clues in detecting their existence:

- 1) Discrepancy of test results with ear pathology or history
- 2) Discrepancy of test results with observable behavior
- 3) Discrepancy of pure tone and speech-hearing tests
- 4) Discrepancy of speech of the individual with extent of hearing loss.

Only one of these centers employed any special test, such as the Doerfler-Stewart Test, specific for psychogenic deafness, routinely on patients, although many of them utilized it and other techniques after the possibility of psychogenic deafness had been raised.

The centers which routinely employed such tests as the D-S Test were those which reported the significantly higher incidence of psychogenic deafness.

In our survey we inquired as to the method or methods used by the center to confirm the possibility of psychogenic deafness once the possibility had been raised. Ninety per cent of the centers reported that they employed repeated testing as a confirmatory check on the original diagnosis. It is our strong feeling that the use of audiometric retests as a method of detecting psychogenic deafness is open to question. The psychogenically deaf individuals whom we have encountered, both in military and civilian centers, have demonstrated their ability, more often than not, to give repeat audiograms well within the limits of error usually ascribed to hearing loss of an organic nature. The remaining centers, while mentioning that they have often found this an unsatisfactory procedure, use psychiatric examination as a confirmation, along with galvanic skin tests.

We should like to list several diagnostic symptoms which we have found useful in alerting our clinicians to the possibility of

psychogenic hearing loss in routine examination of patients. Most of these clues are applicable to the situation of the otologist in private practice.

1) Sudden unexplainable onset of bilateral severe hearing loss, perceptive in nature. It is very rare that psychogenic deafness occurs with good bone conduction.

2) Audiometric slope resembling a saucer, usually lying between 50 and 90 db. The nature of this slope, rising at both ends of the audiogram, has led us to suspect that the psychogenically deaf individual is utilizing, in effect, an equal loudness contour in maintaining the level of hearing which he demonstrates. We have found the "saucer audiogram" to be present in 80% of our psychogenically deaf patients.

3) Unexplainable discrepancy between audiometric results and tests for the hearing of speech. The relationship between these two tests has been well established, and sharp discrepancies, especially where the hearing for speech is better than that for pure tones, should raise one's suspicions as to the organic nature of the loss.

4) Surprising response to therapy out of proportion to what may reasonably be expected. This includes surgical procedures such as fenestration as well as medical treatment. The psychogenically deaf individual is very suggestible.

5) Results of special tests. A number of hearing centers and some otologists employ the D-S Test, which can be given routinely in a matter of a few minutes. Other centers use the galvanic skin response technique both to detect and to confirm the possibility of psychogenic involvement.

SUMMARY

It is our conclusion that there no longer can be any doubt that psychogenic deafness exists in the civilian population in a small, but sizable, percentage. The otologist will find these cases in increasing numbers as he is oriented to techniques in uncovering them.

UNIVERSITY OF PITTSBURGH SCHOOL OF MEDICINE.

XCIV

THE ROLE OF THE OTOLOGIST IN AUDIOLOGY

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Otolaryngology has passed another milestone. Audiology, with all the rehabilitative features which its name implies, is here to stay. Just when we arrived at, and passed, the milestone would be difficult for anyone to state. Perhaps the first audiological clinic ever held was at the session when Eve taught Cain his first word. Phylogenetically, the first clinic was held thousands of years earlier; when the first fully developed cochlea perceived its first sound. Modern audiology, as we know it, however, had its conception at the beginnings of this century. The New York chapter of the League for the Hard of Hearing, certainly one of the earliest centers, has just celebrated its forty-first birthday. In the first quarter of this century, a handful of audiology centers with varying degrees of efficiency was established. But these were only labor pains. Some of the pioneers in this field, who are still with us, would probably agree to this phraseology. The baby was born, after prolonged parturition which began well before World War II, sometime during the early 1940's, and was laid on otolaryngology's doorstep. Without careful nurturing and upbringing it may deny its parenthood and grow up as a kidnapped child. But of this we can be sure, audiology will grow up and become a strong individual part of otology.

If I have seemed to be facetious in my opening remarks, I have not meant to be. Rather I have said what I did with serious intent. Here is a modality in otolaryngology's therapeutic armamentarium, now available, which, if advantageously used, can usefully serve a portion of our patients in an area which previously we have largely neglected. Audiology provides a service to our handicapped patients which, individually, few of us are equipped or inclined to give. It offers, at once, an opportunity and a responsibility to every otolaryn-

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gologist. Individually and collectively we have a role to play in regard to audiology and it is an important one.

Hearing losses have been divided into several categories. For the purpose of our present discussion, the simple two-fold classification of reversible and irreversible hearing losses is convenient. Reversible losses include many conductive lesions and relatively very few losses due to cochlear or nerve lesions. Irreversible losses include the very great majority of all cochlea or nerve lesions and a fair number of irreparable conductive lesions. Audiology is the science of hearing and consequently is concerned not only with normal hearing but with all deviations from the normal. Audiology therefore embraces both reversible and irreversible losses. To say that this turn in events has reached such proportions that there is no need for further work in promoting all that needs doing for people with hearing losses is erroneous. There remains much to be done in establishing proper facilities and in training all otologists, both young and old, for service to these unfortunates. Herein lies the opportunity which audiology offers to otology.

What is this opportunity? It can be general or specific. In general, it offers otologists the chance to support any endeavor which advances rehabilitation of the hearing handicapped. This means we should support national programs which have rehabilitation as their object. We should know about the program of the Conservation of Hearing Committee of the American Academy of Ophthalmology and Otolaryngology and realize that it sponsors state programs for both children and adults. We should know that it has launched upon a far reaching study of the effect of noise on hearing. We should know that it has prepared manuals in the area of rehabilitation which are available to the uninitiated and that it is attempting to improve the standards of audiometry by preparing suitable curriculae for schools of audiometry and creating a registry for audiometric technicians. Its program is far reaching and its interests too many to mention in detail here. We should know about the program of the American Hearing Society; know how it fills a need which is met by no other national organization. We should interest ourselves in a knowledge of what is going on in our own states; what is being done for the deaf and know how well or how poorly it is managed; what the policy is in our states for the detection of hearing losses in school

children and how effectively the needs of these children are met once they are unearthed. We should know something about the problem of noise in industry and the manufacture and sale of hearing aids. And most important of all, we should not confine our interest to a knowledge of these affairs but somehow, somewhere, become involved in them so we can be otologic citizens as well as otologic practitioners.

Specifically, we should support loyally local programs in our own cities and, if there are none, we should be the first to start them. A little over two years ago, according to a survey conducted by the U. S. Public Health Service and the American Hearing Society, there were only 55 hearing centers in the United States. Less than half of them offered all the recognized facilities of such centers. More than half of these centers were located in six states. A number have been established in the past two years but in the aggregate they meet but a part of the need. Who should start such centers in communities where there are none? The otologist. There is a cogent reason why the otologist should be the enthusiasatic initiator of such a center. No center is properly staffed unless an otologist is in charge. Should otologists stand by and watch others initiate such projects and then expect to be invited in to run the show? Hardly. Here is an opportunity for scores of otologists, and they should begin doing something about it at once. The opportunity to guide and lead these new endeavors still exists.

It is appropriate for otology to give leadership in this direction, but we must be equipped to be worthy of that leadership. To be so equipped raises the issue of responsibility. I believe it is our responsibility to know something about the rehabilitative measures which audiology encompasses so we can supervise intelligently the programs with which we are connected. Hence it is our responsibility to know something about lip reading, the trial and fitting of hearing aids, speech audiometry, galvanic skin resistance objective hearing tests, auditory training, the psychology of the deafened and rehabilitative programs for the economically hampered, hearing deficient individual.

Perhaps I can best illustrate the need and wisdom of our acceptance of this responsibility if I address the remainder of my remarks to three aspects of modern hearing centers; speech audiometry, galvanic skin resistance objective hearing testing and auditory training.

The first of these, speech audiometry, has been in use for a longer period than the others, but it would seem fair to say it was not until recent years that it arrived at its present useful state. The study put upon the proper selection of test material by the Psycho-acoustic Laboratory at Harvard, and the practical application of this material by Davis² and others, gave to speech audiometry the lift that it needed to convince many of its practical usefulness. Whether monitored live voice or recorded voice is used for testing, they can be so controlled by electro-acoustic equipment that the tests are accurately reproducible within plus or minus two or three decibels. Excluding the unusual case, speech audiometry thresholds can be correlated to those obtained with pure tone audiometry and thus provide another check on the accuracy of the latter. This relatively new testing procedure relegates to the waste paper basket the whispered and spoken voice tests as they have been used in the past. Several, but notably Glorig, have shown the unreliability of voice tests as used in the average office or as criteria for induction into military service and the evaluation of hearing loss, as a result of such service, in Army, Navy and Veteran Administration centers. It is regrettable that, despite many pleas that they be changed, such tests are still the basis for adjudication of compensation awards for hearing losses among veterans.

In addition to affording another threshold of hearing, speech audiometry through the proper use of discrimination tests provides for one more means of differentiating perceptive and conductive lesions. Admittedly it cannot determine exactly how much conductive and how much perceptive loss there is in a mixed deafness, but what test can? Discrimination tests can be highly predictive as to the usefulness of a hearing aid for the individual tested. A good discrimination score gives reasonable assurance that an aid can be successfully worn. A poor discrimination raises the question as to the advisability of recommending an aid. It should be recognized that there are borderline cases in which discrimination tests are not the final answer. They can give the otologist information, however, which will be of significant help to him in properly advising his patients.

Speech audiometry has proven of worth in testing children who give unsatisfactory pure tone responses and for certain problem adult patients, notably those who show a major discrepancy between hear-

ing for conversation and hearing as suggested by pure tone audiometry.

It has been stated that speech audiometry tests not only the hearing of the patient but his intelligence as well. This is true. But we should not lose sight of the fact that pure tone audiometry does likewise. The part which intellect plays, in comparing the two methods is a matter of small degree. The difference can not be great or there would not be the good correlation between speech and pure tone audiometry which one usually finds. Shambaugh and Carhart¹ have worked out a predictive test for the eventual hearing in a successful fenestration operation, based on bone conduction audiometry. This, they state, has a reasonable degree of reliability. Walsh and Silverman³ have a similar predictive test using speech audiometry for which they claim a like reasonable reliability. What fenestrator will not welcome both and use one as a check against the other, and both as a check against his other tests? Thus, as has been suggested above, speech audiometry provides a comparison of value for pure tone audiometry and, for this reason, should be within the ken of every otologist. Unless we are blind to the errors which arise in our own bailiwick, and thus fail to have tests which are reliable, we will employ all the checks which are available to us. Only in this way will we come close to the true hearing ability of each patient whom we test. If otologists are to be leaders in the new day of audiology they must know something of speech audiometry that they may better discharge their duty to their hearing handicapped patients.

The galvanic skin resistance objective hearing test is a newer audiologic aid than speech audiometry, but it is coming to hold a very important place in the testing of hearing. First presented as a reasonably practical objective hearing test by Bordley⁴ at the New York meeting of this society two years ago, it has been adopted and additionally perfected at several hearing centers. At these it is held to be a medium which fills a long felt need. For reasons not yet determined, the test cannot always be successfully completed, but when successful, it has been demonstrated to have an accuracy beyond initial expectation. Properly conducted, this test has the same reliability that can be expected of any hearing test, which is plus or minus five decibels.

This procedure is invaluable in testing the hearing of the pre-school child. Anxious parents, bringing a child with a suspected

severe hearing loss to a center fortunate enough to have such equipment, can be told whether or not the child hears and how much. Most of us have had to be content to say to such parents as they brought these children to us, that we realized a severe hearing loss existed but we could not accurately measure it. This was always a keen disappointment to the worried parents who were never satisfied. It matters not that the training program to be followed in such instances was much the same as is now given as a result of this new test. What does matter is that with the result of the test before the otologist the whole future educational program of the child can be outlined and his hearing possibilities estimated if the parents follow the advice given. This is a tremendous relief to such parents. More important still, it enlists their immediate cooperation. Without the backing of a reliable hearing test they wonder if the advice given is sound, for there is ever the hope that there is more hearing than is suggested. In such instances they feel they are building a house on sand; that it lacks a solid foundation. Under such circumstances it has taken a good otologic salesman to get his message across. With an objective hearing test at hand, however, parents listen and acquiesce. They know how much hearing their child possesses, they know how large or how small the foundation is upon which they must build, and they begin the program with willingness.

The galvanic skin resistance objective hearing test has another significant value. There is no test which is its equal in detecting psychogenic deafness. The deficit of a psychogenic overlay on an organic deafness can be wiped out with ease. The true state of such a patient's hearing can be ascertained and an accurate appraisal of his future hearing possibilities made. This should be accompanied, of course, with recommendations for appropriate psychologic or psychiatric treatment.

Here is a medium which is a most useful adjuvant and which deserves wider usage than it has at present. It is the obligation of every otologist if he does not wish to bother with the use of this technique in his own office, to see that such equipment is available to him and his patients at some neighboring hearing center, dispensary, or hospital. This is another aspect of the role which the otologist should play in the realm of audiology.

The last of the three specific modalities of modern audiology and the relation of the otologist to it is auditory training. In some

measure this is the least understood of the three. We have been inclined to relegate this phrase of the program to the audiologist and then forget about it. Probably we have done so because in it there is no place for active participation on our part. But just as we have learned that the use of lip reading can improve the communicative ability of the wearer of a hearing aid as much as one-third, so we should know that further improvement in communicative ability is available with the addition of auditory training.

In the time allotted to this presentation, it is impossible to tell the whole story of auditory training. In essence it is to train an individual to use his residual hearing to optimum advantage. It is doubtful if all audiologists would agree how this should be accomplished. The best techniques probably have yet to be evolved. With the methods now available, however, the attempt is made to make the individual listen more critically and to avail himself of all kinds of auditory cues so that proper interpretation may mean increased communicative ability. It is obvious that the individual with a pure conductive deafness does not need the same amount of auditory training which is necessary for the rehabilitation of the person with a cochlear deficiency. Likewise there is less training required for a relatively recent hearing loss compared to that for a long standing unrehabilitated one. An occasional exceptional person can be, (and some are), self trained.

Auditory training serves those with irreversible losses. It has been argued by some that no rehabilitative measures can improve such hearing. Of course they cannot. But they can improve the communicative ability of one who has such a loss. All of the centers which use this training testify to this fact.

Along with auditory training should go appropriate psychologic treatment. For the optimum results there should be rehabilitation of the total person.

The meat of the matter is this. Auditory training is really a necessity for those with irreversible losses whether or not a hearing aid is worn. For those who cannot wear an aid successfully and there are a number of these, it is a real boon. For those who are about to begin the wearing of an aid it is a must. "One of the intangible benefits it affords is the acquaintanceship which the patient acquires with his hearing aid during the supervised early training period. All

the questions which arise regarding this new hearing world are not only answered immediately but are demonstrated in other individuals, so that the first individual realizes that he is not the only one who is having difficulty with his new instrument.²⁵

Unfortunately, auditory training on a first class scale is not universally available. Geographically, its employment is too limited. This is why I have segregated and mentioned it as one of the newer methods in audiology with which the otologist should be familiar. It is a must that he do something about establishing its use if it is not available in his locale. He is derelict in his duty if he fails to do so.

I have outlined briefly the scope and purpose of speech audiometry, galvanic skin resistance, objective hearing testing and auditory training. Otologists cannot wisely advise these diagnostic and training aids unless they know something about them and have made sure they are available for their patient's use. This is argument enough, it seems to me, to show that these are otologic responsibilities.

There is still another phase of the role which the otologist should play which I must mention. It was excellently stated by our president, Kenneth Day, in his Wheery Memorial Lecture last year. I can do no better than reiterate what he said. We should take time to explain to our patients who have an irreversible hearing loss why they have it and why we cannot do anything to improve their hearing. One is amazed at the number of individuals with a hearing handicap who present themselves for consultation and have never had the privilege of such an explanation. There would be considerably less opportunity for the practice of charlatanism among these unfortunates if otology properly shouldered this responsibility.

Much more could be said. There are other phases of audiology in which the otologist has a role to play. But enough has been mentioned to whet the appetite of anyone who has yet to sit down to eat at the table of audiology. All this lacks the excitement of surgery. To many the fun of medicine ends when a correct diagnosis is made. But we have failed to discharge our duty to our patients if we do not make available to them, through our knowledge, all that can bring them closest to their best. This is the heart of the role which the otologist has to play in audiology.

SYRACUSE UNIVERSITY MEDICAL SCHOOL.

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XCV

SUMMATION OF SYMPOSIUM

S. RICHARD SILVERMAN, PH.D.

ST. LOUIS, MO.

I shall have to extemporize this summation since I did not have all of the papers available to me before the meeting.

As the outset I tried to set the stage for this meeting by citing the factors which have influenced the recent upsurge of interest in audiology. I have also tried to indicate that, for many, audiology is an amorphous concept.

Dr. Reger in his presentation made the basic thesis that no equipment is better than the person who uses it, and he preferred that a precise person use simple equipment rather than an unprecise person use precise equipment. He cited the values of speech audiometry but also felt that the tuning forks, pure tone audiometry and even percussion toys, as he calls them, have value; that all of these kinds of apparatus interact with one another to give the total picture.

Dr. Hirsh elaborated the basic concepts involved in the principles of the working of a hearing aid. He clearly delineated the problems associated with attenuation and with the problems of discrimination. His basic thesis was that the attenuation type of deafness could be helped, helped in the sense that more intensity was brought to the ear, whereas one should view with extreme care the recommendation of a hearing aid where the person is suffering from lack of ability to discriminate.

Dr. O'Connor stressed the point that the deaf child does not elicit the sympathetic understanding which the person with a visible handicap does, and he implied, although he did not state specifically, that communication or ability to communicate is at the heart of social adjustment and therefore, in that sense, the deaf child is the most handicapped of all handicapped individuals. He also cited some of the advancements in recent years such as the preschool movements and the attempts to use hearing aids. He also made a stirring plea for your support of the oral method of instruction. He did

not close without a word of warning about the limitations of hearing aids with deaf children. He felt that some individuals and institutions are making unwarranted claims for the possibilities of using hearing aids on deaf children. His basic point was that hearing aids could help the child in the expressive aspect of his communication, in his speech, but it was wishful thinking to expect that the child could learn to communicate by hearing.

Dr. Doerfler stated that psychogenic deafness is a problem. I think that is a significant statement to make, and he indicated that the data which can be relied upon are rather scanty. He felt that the data that he did gather were influenced by the awareness of the individual of the problem of psychogenic deafness. He felt that where more elaborate and sensitive tests for psychogenic deafness were available, the percentage of discovery would probably increase. He closed with some practical suggestions for the otologist in his office for detecting psychogenic deafness.

Dr. Hoople felt that the baby of audiology should not be abandoned by the otologist lest it fall—and I take this by implication—into the hands of charlatans. He made a strong plea for an increasing awareness of the possibilities of audiology on the part of the practicing otologist in the form of general support, specific community endorsement and active participation in audiologic activities. He also stressed the values of speech audiometry, the value of the galvanic skin resistance test, the value of auditory training and the necessity for psychologic treatment. He also tried to get around what he calls the surgical temperament which characterizes your profession, with the hope that one could find a great deal of excitement in dealing with the nonsurgical problems of the ear. I heartily endorse his stand because, if we take a cue from what Dr. O'Connor said, we have in our hands that sense organ which best contributes to the adjustment of individuals. From the papers this morning we see how much we have to learn and we realize that we have in our hands the basic problems of adjustment for thousands, yes, millions of individuals.

CENTRAL INSTITUTE FOR THE DEAF

AND

WASHINGTON UNIVERSITY MEDICAL SCHOOL.

DISCUSSION ON SYMPOSIUM ON AUDIOLOGY

DR. NORTON CANFIELD: Mr. President, Moderator and Guests, I consider that this symposium has presented the essence of conception of the specialty of audiology. Various specialists have presented different aspects of this rapidly developing professional discipline.

We have, however, omitted two important groups. First, the speech specialists, and second, the Social Service workers. We should all understand their contributions, and no symposium is complete without consideration from these two special groups.

Dr. Silverman has spoken of the specialty of audiology as being an "amorphous concept" and one which is still not definite in form. I think he has put it rather gloomily, because it is fairly well defined now. The definition which has been presented indicates the professional phases which should be included. If the definitions already presented are not satisfactory, another one might be added for your consideration. I suggest that audiology is everything that has to do with auditory communication of mental concepts.

We have heard about the various needs of audiology and a number of things should be done. Dr. Hirsh has mentioned the need for another name for hearing aids. This has been discussed on various occasions and some manufacturers are interested. I would like to suggest the word "hearior." Radio and video have been suggested for general use, so "hearior" might be used instead of hearing aid.

To continue the progress of audiology, otology, as it is now organized, has a clear responsibility. Members of this society can immediately carry the work forward on at least five fronts. 1. a popular name for hearing aids is worth considering; 2. appreciation of the problems and potentialities of audiology. This is best accomplished by a continuous self education program of keeping abreast of the current profuse literature on the subject; 3. the dissemination of knowledge and information among your professional colleagues and members of the community. For this, Dr. Hoople has well stated the need; 4. we must encourage younger people to enter any phase of the field so that our slowly growing groups of specialized personnel will be constantly augmented. The 5th need I wish to mention is leadership. Dr. Hoople's discussion emphasized this point extremely well. We have the leaders but unless they are willing to assume greater responsibility, we will soon discover that this segment of the specialty of otology will find expression in other professional channels.

MODERATOR SILVERMAN: I would like to call on some individuals for discussion. Is Dr. Wishart in the audience? Would you like to make any comments?

DR. D. E. STAUNTON WISHART: Mr. President, Mr. Chairman, Gentlemen and Guests, I did not come prepared to say anything. I came to listen, being an otologist who has a vital interest in this problem. My main interest consists in following hard of hearing children in the Hospital for Sick Children, Toronto.

All otologists are aware of the deaf problems in our adult practice. Only too often we find that our patient has an irreversible change in his middle ear or has a perceptive deafness and in many cases it is more or less ridiculous to think we can do anything for the patient. I don't want to raise controversy on that

point but the point that I do want to make is that as otologists we should be chiefly interested in having lesions which cause the great mass of deafness recognized early, recognized as early as possible, and if possible, prevented from becoming irreparable.

I listened intently to hear what audiology might offer otologists who are trying to wrestle with this basic problem. I came interested to hear Dr. O'Connor because I had seen him at the Lexington School, although he has probably forgotten that, but I know that he was very kind to the little people and I hope he has stimulated your interest in what should be done for the children that are deaf.

But once again, gentlemen, that is evasion of the essential point. The children that are deaf are not our basic problem. Unfortunately the mass of otologists are going to be interested in audiology in so far as audiology will enable them to hand over their deaf patients to an organization that will help them find a place in life. Unfortunately Dr. O'Connor passed over the patient the otologist should be interested in but is not—namely, the child that is hard of hearing.

Then I came to listen intently to Dr. Doerfler, because I think probably I am the only Canadian that has a Doerfler-Stewart apparatus. I was interested to hear him say that it had been of use in the testing of children and that there were a certain number of psychogenically deaf little people. So far I have not found any but I am going to be on the watch for them.

Dr. Hoople mentioned the skin galvanometer. We have a skin galvanometer. We are learning what the reactions of normal children may or may not be before we try to make any assertions regarding the hard of hearing children. I don't think that we will be prepared to say anything about it to the Society for the next three years.

Before I sit down, I do hope that some of the succeeding speakers will describe how the new surge for audiology can help the otologist in his hospital and in his office to do more for his hard of hearing patients.

I only have one point to make and that is that little children do not have elaborate hearing tests done by me. In fact, I teach my staff that elaborate hearing tests are useless. What I do want for little children is a measurement of what their hearing is today, done under such circumstances that the test, if repeated six months from now, the same way, will let the otologist know what progress or regression his patient has made.

Thank you very much!

MODERATOR SILVERMAN: Thank you!

I have been asked by the President to call on some other individuals. Dr. Lempert, would you like to make some remarks?

DR. JULIUS LEMPERT: First of all, I wish to congratulate all the participants on this program and the way they presented their part of the symposium. However, I would like to go on record by respectfully stating that for the future good of otology the part of this symposium assigned to Dr. Hoople should have been "The Role of the Audiologist in Otology" and not "The Role of the Otologist in Audiology."

Thank you!

MODERATOR SILVERMAN: I might mention, Dr. Lempert, that before you came into the room, in my introductory remarks I had something to say about that, and

I again must disagree with Dr. Canfield on what he had to imply, when he said I was gloomy. I said that audiology is amorphous.

There is quite a difference between what one sits down and writes about it and what is actually happening, and it is in that sense that I said that audiology was amorphous, not in the sense that we cannot sit down and create a conception. I replied that in my earlier statements I can prove that there are speech correctionists, teachers of the deaf and the whole gamut of these people who are now calling themselves audiologists. I agree with you, however, that it behooves this group, as Dr. Meltzer said in his address last year, to watch this thing with interest.

Dr. Ruëdi, would you like to make some remarks as a disinterested observer?

PROF. DR. RUEDI: I would by far prefer to listen because I think you know more about this than I do.

MODERATOR SILVERMAN: I have one person I would like to call upon and then get back to the volunteers. Professor Muerman is here to study or observe audiological problems. Do you have any remarks?

DR. OTTO MUERMAN (Helsinki): I want to thank Dr. Silverman for his kind suggestion. I should like to say that I am here more for learning purposes than giving any report, but still I am very interested in this discussion and I think that it will be of very big help to me.

I was very much impressed by that which Dr. O'Connor said and what was reported by one of the discussants who was taking part, in that there is a certain difference between deaf and hard of hearing children. I cannot agree that difference exists because in practical clinical experience in Helsinki we have had quite different results than they report.

In Finland for more than one hundred years we have had schools for those that we call deaf mutes but we have not had any school for hard of hearing children, and that is what I am here to study and to try to get started in Finland. I think we cannot mix them now because they have had their experience with the deaf mutes. They know the task. But, on the other hand, of course we may admit that scientifically there is a great deal of difference between the totally and partially deaf but I think we cannot maintain this position from the practical standpoint. I hope that with all the knowledge which I have gathered in the United States, in several centers, I shall have some baggage to begin with and have perhaps then more success when I can explain all these movements you have had in the United States in this respect.

Thank you!

MODERATOR SILVERMAN: Are there any other questions from the audience or comments?

Dr. Goodhill!

DR. VICTOR GOODHILL: Mr. President, Mr. Moderator, and Guests: I thought you might be interested in a report from California on the subject of the word "audiologist." About two months ago Senate Bill 1555 was proposed by one of our state senators, in the California State Senate. This bill was advocated by some hearing aid people for the purpose of the creation of a hearing aid board which would be an autonomous three-man board to control the hearing aid industry of California. The three members of the board would be hearing aid salesmen; no other members were proposed. In the terminology employed in

this official bill, the hearing aid salesmen were to be called "hearing aid consultants" or "audiologists." These are the titles employed throughout the wording of the bill. The bill further stated that an "audiologist" or "hearing aid salesmen" was anyone licensed by the state of California to fit electrical or mechanical contrivances to improve the hearing of the deaf or hard of hearing people.

There was an implication in the wording that legal action would be taken against anyone other than the licensed "audiologist" or "hearing aid salesmen" who would attempt to make such fittings as hearing aids.

This bill came up before the legislative committee in charge of health. Fortunately a few hearing aid men, who were opposed to the bill, notified the Los Angeles Society of Ophthalmology and Otolaryngology. With the cooperation of northern colleagues and the official section of the California Medical Association, the legislators were aptly appraised of the dangerous and unworthy features of the bill. It was consequently tabled in committee and cannot be brought up again for about two years. However, within that period I am certain that innumerable attempts will be made to gain support of otologists and others to put a similar bill before the legislature again.

I thought you might be interested in knowing what is going on in California.

MODERATOR SILVERMAN: I sent a wire on Tuesday to the hearing.

PRESIDENT DAY: The Council acted upon this thing last night.

MODERATOR SILVERMAN: Are there any other comments or questions?

Dr. Fowler, Sr.!

DR. EDMUND P. FOWLER, SR.: I have a copy of this bill which I brought down here to show you what a monstrosity it was. Now that it is squashed we are not so much interested.

There are two aspects of this discussion which I have enjoyed very much and which I think were not brought out sufficiently well. One was the fact that you do not have to just train these children and educate these children, but you have to subject the parents to a series of instructions and exercises so that they can help you. Without the help of the parents you will not get very far, not nearly as far as you will if the parents are properly instructed. Many of these parents are very intelligent people. Many of them are teachers and they can do a tremendous job in helping you teach the very hard of hearing, and the deaf, to hear.

The other point I want to make is that you have at your command, if you will only egg them on, a national organization which will help you establish these centers and who have tried themselves and have in the larger cities established such centers, and this is the American Hearing Society. In the larger cities today they have the apparatus and the personal. They are doing a good job in applying the tests and in guiding these children and properly instructing them in the use of hearing aids and guiding them in their education.

DR. THEODORE E. WALSH: I would like first to offer my congratulations to you, Mr. President, for this most stimulating program.

There are two remarks I would like to make about it. One point was brought up by Doctor Reger when he spoke about the use of the social adequacy index

of hearing in legal cases. I think perhaps the Otological Society is the one organization that could advance this concept, to take the place of "percentage loss of hearing" which, to me, means absolutely nothing. I don't see how one can say that a patient has lost 20, 30 or 40 percent of his hearing and know how well he can communicate; but if you say what the social adequacy index of hearing is, anybody who is familiar with speech testing and the difference between threshold and discrimination can get a perfectly clear idea of what that person can hear in the way of communication.

The other point I wanted to bring up is apropos what Doctor Hoople said. He made a most important point when he said that hard of hearing patients need psychologic help. I think it must be emphasized that the need for psychologic help is from the otologist and is not from the psychologist. You have to be the psychologist with your deaf patients. It is not something that can be done in five minutes or ten minutes, but it takes an hour or more of sympathetic talking and understanding of their problems to help them. Even if you can do nothing medically or surgically for these people, you can give them a terrific lift simply by letting them know you understand their problems and know what they can and cannot hear and by explaining how they can get over their difficulties by a clear admission of their deafness and how they should handle themselves in a crowd.

I think one of the most grateful patients I've had was a girl with nerve deafness for whom I could do nothing but with whom I did spend a great deal of time talking and explaining her condition to herself and to her husband so that they both understood what handicaps she was under.

DR. FRANCIS L. LEDERER: It would be most unfortunate to permit an undercurrent of dissension to interfere with the progress of this work in behalf of total care of the deaf and hard of hearing. There is such a movement now called audiology. I believe in the growth of any field that promises adequate care of the acoustically handicapped. It would be unfair to this movement to want to arrive at a pure definition of audiology. It is not to be construed as representing any one facet of the problem. However, I do not think that it is as "amorphous" as our Moderator would have us believe it to be.

Surely, in the disposition of any given case of severe hearing loss, we are not going to take sides in an argumentative way as to whose job (medical or non-medical) is more important. This Society has set a standard and a pattern for the correct approach to the problems of the hard of hearing and deaf individual. We have represented here surgeons who would have it appear that they are solely interested in the surgical aspects. We have the otolaryngologist who would only feature the medical aspects of the problem. We make a plea for the thesis that somewhere in between the two extremes of thought and action, there must be an area of interest for all of us, whether we are interested in the surgical or medical aspects. This area calls for an understanding of all of the psycho-social-economic aspects of hearing.

The Moderator took the stand that people who sometimes write about the field of audiology do not always practice what they preach. Perhaps, I must confess, that there was much on paper in the otolaryngological past that was not universally practiced, but it has now come into being. I believe we should start from small beginnings in all areas. We must not divorce ourselves from these ancillary groups (the speech correctionist, the psychologist, medico-social worker, acoustic engineer and speech-reading instructor). They are a part of the team

which represents a coordinated therapeutic relationship in behalf of the acoustically handicapped.

I was more than pleased to see this galaxy of Ph.D.'s on the panel because there are certain M.D.'s who would frown upon the Ph.D., and unfortunately vice versa.

A bit of a rumor got around that our Moderator recently told an interesting story. He was under the impression, and perhaps rightfully so, that many of us do a lot of "sniffing around," as he called it. Well, he felt that that "sniffing" was not nearly as important to the patient as is the field represented by the educator. For example, and to illustrate his point, he told a story about a fellow who was caught in a bear trap. While I may not relate it as effectively as he did, it is important for us to remember to place all things in their place. Now, to the man whose foot was caught in a bear trap. Two psychologists—I take it they were Ph.D.'s—came along and saw the man's plight. While they were debating with one another as to what his ids were, and what his family background might be, the man in the meantime was crying for help, a farmer came along. He heard the man's cries and proceeded to get his foot out of the bear trap in a very simple way, without a history, and without an examination. I can relate this story without offense because certainly Dick Silverman is a friend of all of us. I hope he remains that way, because this is no time to divide the camp to argue as to who does "what, with which, and to whom."

I believe that while discussing all of the means of diagnosis and the like, we have forgotten the subject who has lost his hearing, the patient. Dr. Canfield suggested that we bring in several other groups who are interested in the care of this patient. Yes, we could mention some other people who figure importantly in this picture but we must not forget the patient, the one that Dr. Hoople referred to in terms of the *total person*. We disagree very much, and I am sure that we could be supported, that there is such a thing as a psychology of the deaf. The deaf person or the hard of hearing person is what he is because of what he was and where he wants to go. As we leave the subject, may we agree that no operation restores normal hearing, and may we by the same token recognize that no hearing aid restores normal hearing? The otolaryngologist has a complete job to do if he is to carry his patient literally from "the womb to the tomb." This is no area in which he suddenly is finding a need to explore and develop simply because antibiotics have reduced certain of our surgical procedures. Actually, the antimicrobial agents have allowed us to devote the necessary time to more of the fields with which we should be concerned. This is a field that Wendell Phillips and Harold Hayes wrote about in 1922, and I bow with reverence because I used the same title without knowing it, "The Physician's Responsibility to the Deaf and Hard of Hearing." They used the same title 30 years ago, only they were not at that time, in a position to develop it as we are today, fortified as we are with increased knowledge and better instrumentarium, to say nothing of our public health and preventive medicine responsibilities.

This very meeting of these outstanding people in the ancillary groups, meeting as of one mind, promises great developments for audiology, and a very stimulating field for all of us in otology.

Thank you!

MODERATOR SILVERMAN: May I make a remark or two as to the exact nature of the story that I told? Since that was not brought up here this afternoon I will have to discuss that with you on the outside (laughter).

But I have been accused this afternoon of being a pessimist about audiology. I have been by implication accused of rejecting ancillary groups and also by implication of having a Ph.D. I should like to answer the people who have tried to censure me that I hold an academic rank which is Professor of Audiology, so I cannot be very anti.

Secondly, I do hold a Ph.D., and thirdly, I am a member of an ancillary group, so I cannot be ruling those people out.

What I am concerned about are the people who are enthusiastic about audiology being objective and rational about the kinds of things they are trying to do. I certainly am pro and I want that on the record. As for the sniffing and testing children, unfortunately I am in the spot where we get them when they have been sniffed at in dozens of places. They go from place to place but the problem is dropped in our lap and we have the ten year pull. To put it in the language of your profession, we have the major surgery to perform.

The hour is getting late and there are some specific questions we have here. I think some of these can be integrated and one of them concerns the galvanic skin test, its validity and reliability, and it is also related, Dr. Day, to your question on the detection of hearing in children, and then the question, I think from Dr. Fowler, Jr., on the false positives and false negatives in psychogalvanic work.

I wonder if Dr. Bordley would care to make a statement about that.

DR. BORDLEY: Mr. Chairman, Mr. Moderator and Members of this Society: I did not come expecting to discuss psychogalvanic audiometry and I brought no figures with me. In attempting to give you some idea of the work that has been done and some of the conclusions reached, I would like to state that we started this work more than three years ago. We have to date tested more than 700 children. These children are, in the main, pre-school children.

You ask as to the test situation on pre-school children and how valid such tests are. You all realize it is a very difficult thing to take a pre-school child and test it and find hearing loss. We have tested, I think, up to the present time well over 100 children that to all social situations have reacted normally. Those children have given us satisfactory normal threshold responses.

The group we are particularly interested in is composed of children on whom psychogalvanic audiometry was done some three years ago. Such children have now reached the age where we feel by careful subjective audiometry we may have some chance to establish their thresholds. The great majority of the children with that test-retest situation have, so far, averaged out at about a five decibel variation when compared to objective audiometry. Children with that test-retest picture are satisfactory.

We have children, the exact percentage I am unable to state now but I doubt that they run over five or six per cent, that we have been unable to get any satisfactory tests on. They have shown tremendous variation in their responses. They give false positives, if you wish to call them that, and their tests have been very unsatisfactory. Some of these children we now know are children with central nerve pathway lesions. They also have aberrant responses to various other things as they have grown older. One of the tests on which we completely failed was that of an adult. He was a catatonic. He was from a state sanitarium. He was a murderer who had been committed to the institution on a psychiatric diagnosis.

We have yet to get any skin resistance variation on him. We used stimuli so strong that they should have reminded him of the electric chair, but we failed to get any change in skin resistance.

We have seen other children, particularly of the Rh incompatibility group, where there is a tremendous variation in skin resistance response. They have been the most difficult group for us to test. We have tested over 80 of these children. We will probably have to put at least ten of these in the unsatisfactory group.

The more experience we have with this type work the more we realize that in order to get a satisfactory test we must depend upon a team of workers. I believe the greatest difficulty one will encounter is assembling such a trained team to handle these children. The more you work with children of the pre-school group the more you have to learn about their handling, about giving them confidence and about not hurting them. It is to be years before any of us will have a sufficiently well trained team to be able to accurately know all the things we should know about psychogalvanic skin resistance testing. Such audiometry certainly is not perfect. It has possibilities.

I was very interested in Dr. Wishart's statement that it would be three years before his group would report their work. I applaud him because I think that he is working with a group up there that has taken great pains to find out how we tried to set up our clinic. I feel that they are going to come out with some of the really essential answers that are necessary in this work with children.

Thank you very much!

MODERATOR SILVERMAN: Thank you!

I am going to try to wrap up some of these other questions. This is a question addressed to Dr. Reger. "Do you think that speech audiometry in its present form is as accurate and as useful to the average otologist as is pure toned audiometry?"

DR. REGER: I am tempted to say yes it can be in its present form if the average otologist will study it and know how to employ speech audiometry. I assume the average otologist would still have the tuning forks, possibly for checking results and for bone conduction measurements, if he likes, or bone attachment with the speech audiometer.

MODERATOR SILVERMAN: The next question is addressed to Dr. Doerfler. I think he can answer a couple of questions. 1. "What is this test of yours?" 2. "How about false positives and false negatives in your test and what causes the misses, if any?"

DR. DOERFLER: The principle is rather simple. If we assume that an individual has normal hearing and his body is entertaining a hearing loss, he must carry around with him a yardstick whereby he can reproduce this hearing loss. The way in which normal people gauge the loudness of any sound is to gauge it against the background in which it occurs, quite reasonable noise such as we have in this room and so forth. Therefore, if we destroy the reference level which the psychogenically deaf person is using to reproduce this hearing loss, we make it relatively impossible for him to be consistent in his maintenance of a hearing loss of this level. That in brief is the principle of the D.S. test.

We introduce a noise background while the individual is repeating words at a level above his avowed threshold. This noise destroys the reference level and he gives, shall we say, positive results. I have seen quite a few centers using this test very much like the galvanic skin test. It is open to variations in its use and the results will vary in terms of the use and the manner in which it is used.

If the test is used in the manner in which we use it, I think that it will prove very useful in picking up the psychogenically deaf individuals.

On those cases where we get positive results we have always found either an exaggeration of an organic loss, a pure psychogenic loss or a few associated conditions such as head injury and the like. Where we have obtained a negative result we have not yet demonstrated the presence of psychogenic deafness.

DR. LURIE: Describe the test.

MODERATOR SILVERMAN: Describe the test on the patient.

DR. DOERFLER: It involves the same apparatus that Dr. Reger described for speech. The individual's threshold of perception for certain spondee words is obtained. The level of presentation is then raised five db. At that level when we feed him words, he will repeat these words almost 100 per cent of the time, while these words are going into both ears through headphones. Usually it can be done binaurally while he is repeating them. A noise background, usually a type of noise described as saw-tooth with a base frequently of about 125 cycles, is brought in through the headphones at a gradually increasing intensity. He is still repeating these words. A negative test, and it varies with the type of noise, is found when the noise background has to be louder than the presentation of speech before he stops repeating the words. The psychogenically deaf individual will characteristically stop repeating the words at a level where the noise is fainter than the speech, far below where it should interfere with his ability to hear and repeat these spondee words if his hearing loss were organic in nature.

I should mention that it is a useful test for the malingerer.

DR. EDMUND P. FOWLER, JR.: If you use a low pitch tone primarily, don't you get a diagnosis of psychogenic deafness ipso facto because you are not using high tones for your masking noise?

DR. DOERFLER: The test does not—and I refer you to the article in the *Journal of Speech Disorders*, Vol. 11, which describes it—pick out the psychogenically deaf individuals. It picks out a group of individuals about whom some suspicion should be raised, that they may have a history of head injury, psychogenic loss and other conditions. So often you get a positive test when your history tells you that the diagnosis is.

We have tried using various types of noise such as thermal noise. We don't find them as effective. Other institutions have been using thermal and square wave noise.

MODERATOR SILVERMAN: A question is raised, directed at Dr. Doerfler or anyone else around here, concerning his opinion on the use of electro-encephalography to detect hearing in infants and in suspected malingerers?

DR. DOERFLER: The University of Illinois, as most of you know, has done much of the work with brain waves and hearing loss. I should just like to say it offers a great deal of promise but it is an unwieldy instrument to use clinically.

A lot more has to be done both in the refinement of the diagnosis and in the stimuli used before we have definitive answers.

MODERATOR SILVERMAN: We are coming to the end of this. There is one question that Dr. Berry asked about the otologist selecting or suggesting, recommending hearing aids in daily practice without the availability of any kind of elaborate apparatus.

I think Dr. Hirsh touched on the principles of that and my own feeling is that having established at least to your own satisfaction the difference between attenuation deafness or conductive deafness and the other type, that one can guide himself accordingly. I feel that some of these principles can be applied in the office.

For example, the very simple question or simple comment of the patient who says, "Doctor, will you speak louder? I cannot hear you," or the patient who reports, "I hear people but cannot make out what they are saying," obviously the first one is a candidate for a hearing aid without much more measurement. I also feel that one can get some of these words, be they nonsense syllables or PB words, whatever words you choose, and even though you do not have accurate measuring equipment, I don't think there is anything to keep you from turning the patient around to eliminate lip reading and repeating some of the words and see how well he does. I am aware that this does not meet all the rigorous scientific criteria but I think it can be extremely useful in office practice. As a matter of fact, speech audiometry as it has been described here this afternoon is merely an attempt to quantify that basic concept. That is all it is, and as Dr. Hoople said, I think the statement that escaped us, speech audiometry preceded most of the audiometry and all that we tried to do was to quantify, and in the hands of the skilled otologist who is used to dealing with these problems, I have full confidence that he can make a good recommendation without any quantitative measures. I don't recommend it but in the practical situation I feel that can be done, since I disagreed with Dr. Canfield before. I should like to close this summary. Is there any comment, other comment? Perhaps the essayists have something to say before I summarize?

Dr. O'Connor?

DR. O'CONNOR: No.

MODERATOR SILVERMAN: Dr. Hirsh has left. Dr. Hoople, do you want to say something on this?

DR. HOOPLE: I would like to say two or three words, one about the skin galvanometric testing. I made a statement as I spoke that it cannot be used successfully in every case and I hope that was understood, even though I spoke enthusiastically about it. Our percentage in Syracuse is not as high as Dr. Bordley's. Probably that is because we have not yet developed as good a team as he has. However, if only one test in ten is successful it is worthwhile, not only from the standpoint of the fact that you have an audiometric reading of the child, but from the standpoint of the satisfaction it gives the parents. They know how much hearing their child has and that makes a profound impression on them.

If in your office you tell parents their child has a profound loss but you cannot tell them exactly how much, it leaves a suspicion in the parents minds that you don't know what you are talking about and there is more hearing present than you have indicated because they have the hope—hope springs eternal—there is more hearing in the child than really exists. I urge you to get interested in

this, knowing full well it is beset with difficulties and that it cannot be used successfully in every case, because it will prove tremendously worthwhile to you if you care to work with it.

The other thing I want to say, is to amplify, and if I may be presumptuous, to correct a statement of Dr. Wishart's. He said he would like to learn from this discussion what can be practical to the otolaryngologist in his office because after the otolaryngologist has made the diagnosis of an irreversible hearing loss he must hand his patient over to an organization which will take over the problem. I would like to stress the point that after a diagnosis of an irreversible loss is made the patient should be handed over to AN ORGANIZATION IN WHICH THE PRACTICING OTOLARYNGOLOGIST HAS AN ACTIVE AND INTERESTED PART. I have time and again referred such patients to our hearing center for the fitting of a hearing aid, auditory training or the like. When I have gone to the hearing center to visit it during clinic hours and sessions and they have seen me there, it has tied the whole thing into one package as far as the patient is concerned. If I am there and they do not see me, they feel I am not interested in them and the hearing center is a separate organization for them.

To the question that several have raised as to the part that the audiologist has to play in otology, I would say that trying to separate the two is silly. We need to be interested in audiology from the standpoint of our patients, for the good we can do them by our participation in it because what they want is the doctor's opinion. Our society is geared today, (whether or not you wish to have it otherwise you cannot change it), so that when a patient has a physical handicap he wants the doctor's opinion as to its severity and prognosis. If the doctor is not in on this program, enthusiastically supporting it wherever he may be located, he is denying his patient something that he desperately desires.

MODERATOR SILVERMAN: Before I summarize the summary, Dr. Day has a comment he would like to make.

PRESIDENT DAY: The one comment I have to make is, I think there is one feature of this tremendous subject which has been passed over too lightly. Stressing of the training of the pre-school child and its parents is the most appealing program we have for the public. Remember that this is part of audiology.

In the last two years they have been holding classes at the University of Pittsburgh for the parents of hard of hearing children. We started with a small group in the first class, with ten or twelve lectures. The class has been repeated how many times, Lee?

DR. DOERFLER: It has been continuous since 1948.

PRESIDENT DAY: When you find a registration of over 100 people trying to come in to a class to learn about the children, then we begin to see what active public interest there is. That has just gone over like a million dollars with the public of our city. To get these little children in the preschool age and get their training started, that is the place on which you want to put most of your stress and that appeals to the public and after all, if we are going to develop audiology centers, we must consider the public appeal because they will provide the funds.

MODERATOR SILVERMAN: In summary, just a minute or two, as I started to say, as I had disagreed with Dr. Canfield before, I should, now want to agree with him, perhaps as the nub of this whole symposium, when he said that in this

group lie the leaders of this movement and those of us who are in audiology or ancillary or auxiliary troops, would rather do away with the notion that this is an integrated attack. This is the senior society and the problem is not advanced or brought nearer to solution by turning up one's nose at it. If one has any criticism of it he should try and participate in it, so—and I speak only as an individual, I cannot speak for the profession of audiology—we look to you, to your skills and to your inspiration for cooperation and leadership.

THE DEVELOPMENT OF THE OTIC CAPSULE IN THE
REGION OF THE VESTIBULAR AQUEDUCT

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AND

THEODORE H. BAST, PH.D.

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Several aspects of the authors' inclusive study have already been reported at successive annual meetings of the American Otological Society and subsequently recorded in a series of articles in this journal.¹⁻⁶ They were concerned with the developmental and adult anatomy of the auditory ossicles, of the otic capsule and extracapsular tissues, of the endolymphatic sac, and of the capsular wall of the lateral semicircular canal in the region of surgical fenestration. In the present article, the authors will review the major features in the morphogenesis of another special portion of the otic capsule. The observations herein recorded are based upon an examination of a large number of series of sections in the otological collection at the University of Wisconsin. Representative specimens, five in number, were selected for illustration, in figures which will serve as the chief bases for the observations recorded in the text.

The writers have already established the fact that maturity in gross form and size and in histological structure is attained by the otic capsule, as well as by the auditory ossicles and membranous labyrinth, far earlier than was once believed, and in ways that mark it as the most highly specialized element of the human skeleton.

From the Department of Anatomy of Northwestern University Medical School (Contribution No. 571) and the Department of Anatomy of the University of Wisconsin.

Paper read at the Eighty-fourth Annual Meeting of the American Otological Society, Atlantic City, May 11-12, 1951, and at the Sixty-fourth Annual Session of the American Association of Anatomists, Detroit, March 21-23, 1951.

This study was conducted under the auspices of the Central Bureau of Research of the American Otological Society.

As pointed out in antecedent articles, in the surgically important region of the otic capsule, that is, the tympanic wall of the lateral semicircular canal, the process of ossification is, in its early stages, essentially similar to that which takes place in any typical portion of the capsule. However, at approximately the stage of midterm, the process of ossification in the region of surgical fenestration begins to follow a special course. Within a period of four weeks thereafter, the recently formed outer (periosteal) layer has been largely removed. So rapid is this process of resorption that soon varying amounts of the middle layer (endochondral bone and the cartilage islands) are likewise destroyed. Subsequent to this stage of resorption, during the course of which tympanic air-cells are abortively formed, new bone is added on the tympanic surface, to restore a smooth contour. As a result, the mucoperiosteal membrane covers this secondary bone, instead of investing an original periosteal layer. Thereby, the tympanic wall of the lateral canal, after having been eroded deeply on the external aspect, is rebuilt to assume regular thickness.

In the current report, consideration will be given to the development and adult structure of the opposite (meningeal) wall of the capsule, in the territory of the cranial orifice of the vestibular aqueduct and in the adjacent portion of the posterior surface of the petrous pyramid. Rarely the site of pathological tissues, this area undergoes a series of morphogenetic changes, resembling those above-mentioned, by which the tympanic wall of the lateral semicircular canal is formed, then resorbed, and finally rebuilt.

OBSERVATIONS AND DISCUSSION

The otic capsule differs from all other skeletal elements of the human body in respect to function, rapidity of development, mode of ossification, and histological fabric. Actually, in every phase of their otological investigation, the authors have been surprised both by the unique character of capsular structure and by the manner of its genesis.

The development of the capsule is rapid; it attains adult dimensions when the fetus reaches the middle of its intra-uterine existence. In contrast, a typical long bone continues to grow through a period of 20 or more years. Although the otic capsule retains fetal dimensions throughout life, it does acquire a thick investment of extra-

capsular periosteal bone, and, as a consequence, becomes the labyrinthine core of the so-called petrous part of the temporal bone. The otic capsule is unique, too, in being formed from 14 primordially independent ossification centers; bone spreads almost simultaneously from each of these originally separate centers, not, as in a typical long bone, from a point of initial ossification in the middle of a diaphysis toward terminal epiphyses. Fusion between centers is peripheral and complete; there exist no zones of secondary, or epiphyseal growth such as regularly occur in a long bone. Fusion, with complete erasure of fusion-lines between contiguous centers, early converts the otic capsule into an osseous unit. Histologically, too, the otic capsule is exceptional. In the antral region of the capsule, the outer one of the three capsular layers becomes pneumatized through invasion by mucous membrane. The middle layer, most complex of the three, retains throughout life a considerable fraction of its primitive but calcified cartilage matrix in the form of intrachondral bone lodged in endochondral bone. This layer of dual origin is never converted into haversian bone. The inner capsular layer forms a thin, unchanging shell for the labyrinthine canals, the cochlea and the vestibule; it fuses with the endochondral bone of the middle layer.

The periosteal (or external periosteal) layer forms a complete outer shell earlier in the cochlear than in the canalicular division of the capsule. This outer layer is destined to produce all of the extracapsular tissue; as a result of its thickening, the adult dimensions of the temporal bone will be attained. While its bulk is being thus increased, the original labyrinthine capsule (bounded by the endosteal layer) undergoes no enlargement; its fetal size is maintained unaltered in the adult.

The endosteal (or internal periosteal) layer, produced by the periosteum on the inner surface of each of the several ossification centers, forms a thin wall of the labyrinthine spaces. Production of this layer is begun in the fetus of 150 mm (11 weeks); it is complete within a three week period (in the 183-mm, 21-week, stage), except where it constitutes the peripheral portions of the semicircular canals.

Intrachondral bone appears before endochondral bone in the middle layer. The former type of osseous tissue is present in the fetus of 120 mm (16 weeks); attaining maximum development in



Fig. 1.—Photomicrograph of a transverse section through the otic capsule at the level of the cranial orifice of the vestibular aqueduct in a fetus of 150 mm CR length (18½ weeks). Wisconsin series 39, slide 17, section 6. X 18.

On the medial wall of the cranial orifice, the cartilage is still unmodified; on the lateral wall, ossification has been initiated (forming Center 10 of Bast's description). In this area the cartilage is being converted into intrachondral bone; in later stages in the process of ossification, the primordial cartilage will persist, to appear as islands of modified cartilage in a field of mature bone. In the region illustrated, neither periosteal (outer periosteal) nor endosteal (inner periosteal) bone has yet formed.

the fetus of 183 mm (21 weeks), the intrachondral bone (cartilage islands) never wholly disappears. Although in some regions of the capsule the cartilage islands may be destroyed, in other parts they seem to be as abundant in the adult temporal bone as they were in that of the midterm fetus. Soon after the endochondral bone attains widespread distribution in the capsule, the formation of endochondral bone is initiated. The latter is deposited upon the islands of calcified cartilage.

In the region of the cranial orifice of the vestibular aqueduct and in that of the adjacent fovea of the otic (endolymphatic) sac, formation of an outer layer of bone is delayed. To the morphogenesis of this part of the otic capsule, the discussion will now be devoted.

In the 50-mm (ten week) fetus, the otic capsule has but recently become a definitely bordered cartilaginous unit. The course of the vestibular aqueduct at this stage predicts that of the channel in older specimens; the aqueduct passes obliquely from a vestibular

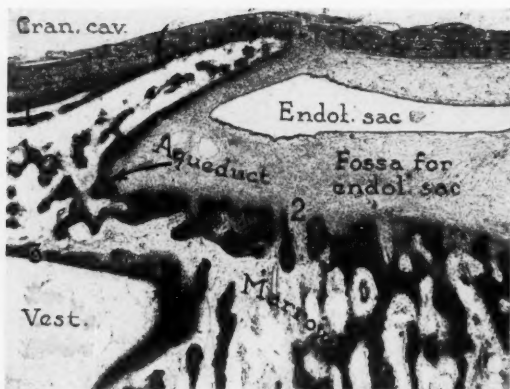


Fig. 2.—Photomicrograph of a transverse section through the capsule at the level of the cranial (dural) aperture of the vestibular aqueduct in a fetus of 183 mm (21 wks., approximately midterm). Wis. ser. 21, sl. 26, sect. 1. X 18.

In the area of the ossification center (compare Fig. 1), endosteal bone is being deposited upon the islands of cartilage; the combined tissue constitutes the middle layer (layer 2) of the otic capsule, which here, not yet being covered by periosteal bone, is exposed to the dura mater in the fovea for the otic (endolymphatic) sac. Periosteal bone (at 1) is present as a thin plate on the medial (cranial) surface of the ledge (as far as the unnumbered arrow). The orifice of the aqueduct, early formed in cartilage, now consists of osseous walls and a core of sparsely distributed intrachondral bone. Endosteal bone (at 3), forming the wall of the vestibule (as it serves, also, for other portions of the labyrinth), has already become fused with the adjacent portion of the middle layer.

to a cranial aperture. From the cochlear portion of the capsule, the prominent medial wall extends as a short, yet relatively bulky, ledge.

In the 100 mm (14-week) fetus, the cartilaginous capsule is appreciably larger than it was in the preceding stage. With general growth has come marked elongation of the ledge-like medial wall of the vestibular aqueduct.

In the fetus of 150 mm (18½ weeks) an ossification center has appeared on the lateral wall of the vestibular aqueduct (Fig. 1). Within the ossifying area, intrachondral bone is being formed, as the primary tissue of the middle layer; neither periosteal nor endosteal bone has yet appeared, and the medial wall of the orifice of the aqueduct is still cartilaginous.

The second important step in the process of ossification of the vestibular aqueduct is evidenced in the fetus of 183 mm (21 weeks). Through spreading ossification, the ledge which guards the cranial

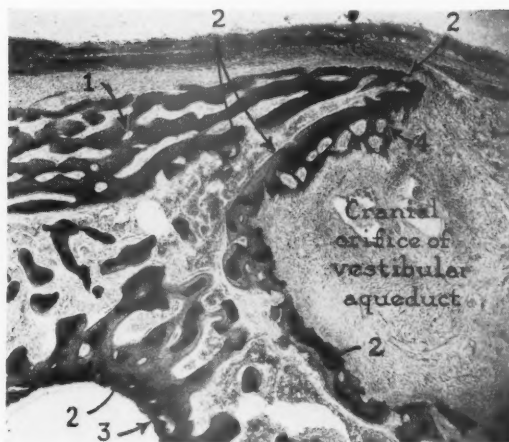


Fig. 3.—Photomicrograph of a transverse section through the otic capsule at a level of the dural (cranial) opening of the aqueductus vestibuli in a 215-mm (24-wk.) fetus. Wis. ser. 62, sl. 13, sect. 5. X 18.

The outer, periosteal, layer (at 1) has undergone considerable thickening. On the wall of the vestibule, the inner, endosteal layer (at 3) is fused with the middle layer of the capsule. The middle layer (at 2) is exposed to the dura mater within the cranial orifice of the aqueduct and along the wall of the fovea for the otic sac; it is likewise uncovered on the medial surface of the ledge. Not only has the middle layer not become wholly covered by periosteal bone (outer layer), but actually is being eroded. As a consequence, intrachondral bone, once invested by endochondral bone, is now in contact with meningeal tissue. Concurrently, the lateral wall of the ledge is receiving a new covering; here membrane bone (at 4) is being deposited upon the formerly irregular surface of the middle layer.

orifice of the aqueduct now consists of the three typical layers of bone (Fig. 2). On the medial wall, the periosteal layer is still thin, as is also the endosteal where the latter bounds the vestibule. Between these strata, the middle layer consists of sparsely distributed intrachondral bone. Proximal to the point at which the ledge becomes continuous (Fig. 2, at unnumbered arrow) with the fovea for the otic (endolymphatic) sac, the outer layer ends, to leave the middle layer to the dura mater. The middle layer, except for that part of it which forms the core of the ledge, no longer consists solely of intrachondral bone; upon the islands endochondral bone is being deposited. Ultimately, through continued production of the latter, the intervening marrow spaces will be reduced to small spaces for transmission of the blood-vessels. Generally speaking, the intrachondral bone remains relatively unaltered throughout life; how-

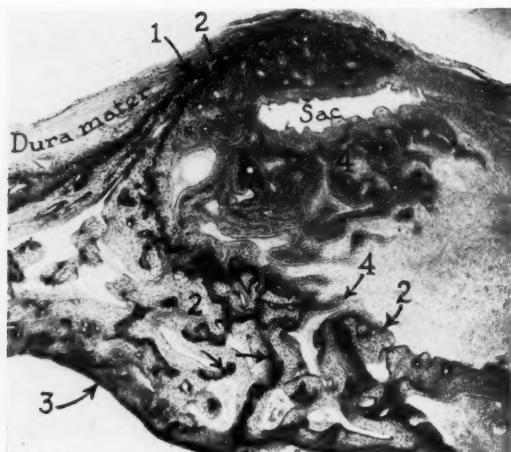


Fig. 4.—Photomicrograph of a transverse section through the orifice of the vestibular aqueduct in a fetus of 270 mm (30 wks.). Wis. ser. 107, sl. 13, sect. 8. X 18.

The ledge of bone has undergone further alteration in form through the production of additional membrane bone (at 4). The original periosteal part remains as the investment of a slender projection (at 1). Within the orifice, membrane bone has enveloped the otic sac, to produce a new, or secondary, cranial opening for the vestibular aqueduct. This layer is spreading along the medial wall, covering bone of the middle layer as it progresses. In the middle layer, the persistent intrachondral islands remain (as at the unnumbered arrows) to become permanent adult constituents of the capsule.

ever, in the core of the ledge, the islands of intrachondral bone are destined to be destroyed in later developmental stages.

In the fetus of 215 mm (24 weeks) the periosteal layer on the medial (cranial) aspect of the ledge is considerably thicker than it was in the fetus of 183 mm; the layer gradually thins and finally ends at the tip of the ledge (Fig. 3). Within the core of the ledge, the middle layer is now almost devoid of intrachondral islands; however, in the two remaining thin strata of endochondral bone (at double arrows in Fig. 3) a few such islands remain. On the lateral aspect of the ledge, one phase of the third in the series of developmental steps is being evidenced: membrane bone is being deposited upon the remnant tissue of the middle layer. Another phase is represented by the erosion of the lateral wall of the fovea and aqueduct; removal of this middle layer includes destruction of the intrachondral bone. Through the operation of these concurrent

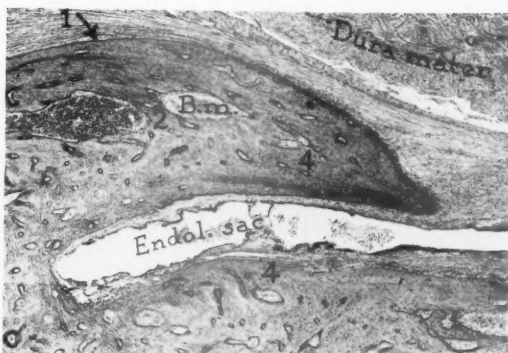


Fig. 5.—Photomicrograph of a transverse section through the capsule at the level of the cranial orifice of the aqueduct in an infant of 10 weeks. Wis. ser. 83, sl. 46, sect. 2. X 18.

Periosteal bone (at 1) forms the outer layer of the ledge; the more recently formed membrane bone (at 4) has spread beyond the point reached in the 270-mm fetus. The new bone lines the entire aqueduct and virtually all of the fovea. The middle layer (at 2) here remains only as the core of the ledge. The orifice of the aqueduct is but little larger than the contained endolymphatic sac. Abbrev., *B.m.*, bone marrow.

processes of deposition and erosion, the cranial orifice of the aqueduct and the fovea of the endolymphatic sac are altered in form and size.

In the fetus of 270 mm (30 weeks) the ledge of bone which forms the medial wall of the orifice, at first seemingly compressed and composed almost wholly of endochondral bone, is now lengthened by the addition of membrane bone, which forms within the previously excavated, enlarged, fovea (Fig. 4). Not only has the new bone served to store the length of the ledge, but acts also to enclose the otic sac in a less capacious fovea. The membrane bone will continue to spread along the floor of the fovea. The bone of the middle layer, other than that of the ledge, contains prominent islands of intrachondral bone, lodged within endochondral bone. Enlargement of the endochondral spicules has been sufficient to reduce the dimensions of the marrow spaces to channels but slightly more capacious than the blood-vessels which they transmit. The inner (endosteal) layer is not as clearly distinguishable from the middle layer as is the newly-formed membrane bone.

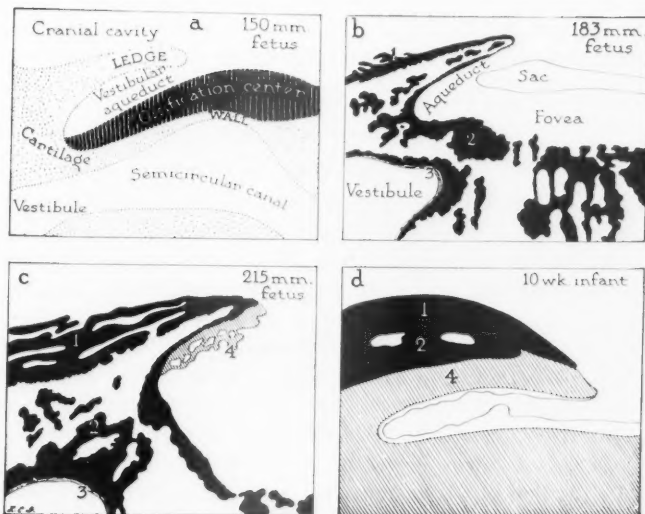
Formation of membrane bone within the connective tissue of the orifice and fovea continues until the ledge consists chiefly of that

tissue. The result of this alteration is shown in the ten-week infant (Fig. 5). The aperture is narrowed, and almost completely filled by the otic sac. On the medial wall of the ledge, the periosteal layer ceases to be readily distinguishable from the original endochondral bone. The same is true of the lateral surface, where the preexisting bone can no longer be distinguished from the newly-formed membrane bone. Thus, ultimately, both walls of the aqueduct attain the three-layered structure.

CONCLUSIONS

The otic capsule is unique among skeletal elements in respect to several important features, which concern both the morphogenesis of its parts and the character of its constituent tissues. Since some of these features affect the special region of the vestibular aqueduct as well as the capsule as a whole, they may properly be reviewed here, in introduction to the presentations of the authors' current conclusions.

Developmentally, the capsule represents a fusion of numerous, originally independent, ossification centers. At the time of fusion of the contiguous centers (in the middle of intrauterine life), the labyrinthine spaces thereby enclosed by a thin stratum of bone derived from the inner periosteum, will have attained adult dimensions. Subsequent activity of the outer periosteum, while serving grossly to imbed the primordial capsule in the petrous part of the temporal bone, leaves the size of the labyrinth unchanged. The inner periosteal (endosteal) layer is unlike the corresponding stratum of a typical long bone in ceasing to be active in the midterm fetus. In addition, its relationships are of a special order, since it encapsulates a system of interconnected epithelial tubes—cochlear, utriculosaccular and canalicular. The middle layer of the capsule is also unusual, but in still a different way: structurally, it consists of two dissimilar types of bone, whose initial appearance is not simultaneous and whose periods of maximum growth are not concurrent. Of these two, the tissue consisting of islands of ossifying cartilage is the first to appear; production of endochondral bone soon follows, the latter being deposited upon the cartilage islands (inrachondral bone), to imbed them in spicules. Thickening of endochondral covering, in correspondingly reducing intervening marrow spaces, accounts for the petrous nature of otic capsule. Once formed, the histologic pattern of the middle layer never changes in normal bone; the combined



Figs. 6a to 6d.—Four crucial stages in the development of the otic capsule, respectively, of fetuses of 150 mm, 183 mm, 215 mm and infant of 10 weeks. Schematized from figures 1, 2, 3 and 5.

intrachondral and endochondral bone is never replaced by bone of haversian type.

The series of developmental steps through which the capsule passes in attaining mature form, size and fabric is, to a degree, followed by the capsular wall in the region of the vestibular aqueduct and of the adjacent fovea for the otic (endolymphatic) sac. However, in this territory of the cranial aperture of the vestibular aqueduct and neighboring foveate excavation, the series of changes in the constituent layers depart even more strikingly from the standard pattern of genesis. These steps may be traced in the following selected stages. Late production of an outer layer of bone in this zone is here the result of change in plane of the endolymphatic duct and the enlargement of the endolymphatic sac. The histological replacement of some of the tissue of the middle layer is the result of the same causes. These processes effect the structure of the ledge which, medially, intervenes between the general space of the cranial cavity and the aperture of the vestibular aqueduct, and, concur-

rently, the structure of the wall which, laterally, intervenes between the aqueduct and the vestibule. They may be summarized as follows, in reference to schematized steps (Fig. 6).

In the fetus of 18 to 19 weeks (150 mm) the ledge is still completely cartilaginous; on the lateral wall of the aqueduct, an ossification center has appeared (Fig. 6a).

Within a three week period (in the fetus of 21 weeks, 183 mm) the process of ossification has progressed to the point at which the three typical layers have appeared in the greater area of the capsule (Fig. 6b). However, in the region of the aqueduct, although periosteal bone forms the medial surface of the ledge, bone of endochondral and intrachondral types constitutes the opposite wall. The same middle layer is continued, denuded, along the fovea for the otic sac. The endosteal, or inner periosteal layer, is now not only well established, but has virtually attained adult character. Forming the wall of the periotic labyrinth, it will be subjected to neither marked histological alteration as a layer nor change in area as the wall of labyrinthic spaces. However, in some specimens the layer fuses so completely with the endochondral bone of the middle layer that the line of original demarcation between them is wholly lost.

Within another three week period (in the fetus of 24 weeks or 215-mm), striking changes have occurred in both surfaces of the ledge and along the surface of the fovea (Fig. 6c). The substance of the original ledge has been almost completely destroyed and replaced by bone marrow; the remnants thereof, which do persist, appear as two thin, facing, laminae in which a few cartilage islands are discernible. The medial surface of the ledge is covered by a thick periosteal layer; whereas membrane bone is being deposited on the lateral surface. Along the remainder of the wall of the cranial aperture of the vestibular aqueduct, and continuously into the fovea, the middle layer of bone remains denuded, being exposed to the dura in which the otic sac is lodged. Here, erosion rather than deposition of bone is occurring; recently exposed endochondral bone has been destroyed, as have also some of the contained cartilage islands, in evidence of a process which transitorily deepens the fovea.

In early postnatal stages (for example, in the ten-week infant), the form and size of the aqueduct and fovea have been profoundly altered (Fig. 6d): the cranial orifice of the aqueduct, now a crevice

(as seen in horizontal sections) is only slightly more capacious than the sac which it contains; the fovea is shallow, that is, not the excavated depression which it was in the temporal bone of the 24 week fetus. The ledge, whose thickening has taken place at expense of the space over which it projects, now consists of three layers, namely, a relatively thin periosteal layer, an endochondral layer and a thick stratum of membrane bone. A relatively considerable distance now separates the middle layer of the capsule from the space of the orifice of the aqueduct; this distance, which is the thickness of the new membrane bone, represents the former depth of the excavated fovea. Despite the fact that such dramatic modifications have been made in the aqueduct and the fovea, the layer of endosteal (internal periosteal) bone remains unchanged. Rebuilding of the aqueduct, like that earlier described for the tympanic wall of the lateral semicircular canal, in no manner affects the structure of the layer which, as the innermost of the three osseous laminae, forms the boundry of the labyrinthine (periotic) system of spaces.

It is because the "adult" position of duct and sac is assumed and mature curvature established in the infant, not in the fetus, that remodelling of the aqueduct and fovea must take place in bone, not in cartilage. A very different series of events accompanies, or rather, permits, enlargement of the arcs of the semicircular ducts: cartilage is removed peripherally to permit of their advance; new tissue is concurrently produced to maintain the canalicular configuration of the surrounding periotic spaces. The process is aided by the rapid excavation of cartilage in the subarcuate fossa.

Thus it is that two neighboring areas of the otic capsule follow markedly different patterns of development. Neither of these schemes of reconstruction is comparable to the excessively slow series of structural changes which occur, in many specimens, in the region of the fissula ante fenestram. Although the fissula is formed so early that its typical form, contents and parietal layers are established in later life, histological instability seems to characterize the region in postnatal stages. Either within the fissular channel, or in tissues which immediately bound the latter, cartilage and bone retain power of postnatal activity with such frequency that the region has long been recognized as the site of predilection for otosclerosis. These observations lead inescapably to the conclusion that the late attainment of adulthood by

tissues of the capsule does not necessarily make for malleability; the observations further suggest that other factors must be operative in the local production of bone of otosclerotic type.

303 E. CHICAGO AVENUE.

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XCVII

ANTI ALLERGIC METHODS USED IN THE RESTORATION
OF HEARING DURING CHILDHOOD

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RICHARD J. BELLUCCI, M.D. (By Invitation)

AND

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NEW YORK, N. Y.

The experience of recent years has proved that allergy is an increasingly important cause of many nose and throat diseases. We are convinced that allergy also affects the ear and can cause impaired hearing. Hearing loss may be prevented, restored to normal or improved by judicious anti-allergic treatment.

During the past two and one half years the Conservation of Hearing Clinic, connected with the Manhattan Eye, Ear and Throat Hospital, has conducted a study of impaired hearing during childhood. Previously one of us (Jones) had observed hearing improvement as the result of treatment for allergy.¹ Our present study includes all causes and treatment of impaired hearing in children. Perhaps the greatest stimulation to organize such a program on a broad basis was the report by Crowe² of encouraging results following radiation of adenoid tissue. That work was done in an unusually meticulous and laudable manner. Such accurate data for estimating hearing function were impossible to obtain before the perfection of the audiometer. We determined to re-examine the effects on hearing of tonsillectomy, adenoidectomy, sinus surgery and palliative treatment of the sinuses,

From the Research Department of the Conservation of Hearing Clinic, Manhattan Eye, Ear and Throat Hospital.

This work has been partially subsidized by and is under the auspices of the Central Bureau of Research of the American Otological Society.

using modern methods. In addition, we included a more recent concept of the cause of hearing impairment allergy.

In order to avoid confusion and at the same time admit some of our handicaps, we will mention the conditions under which our work has been done.

The testing rooms are sound conditioned at an acceptable and reasonably constant level. Absolute sound proofing against the external sounds of New York was not feasible.

We have found the zero line of our audiometric charts does not represent the average normal of the patients we have tested. We have been experimenting with a recorded range which starts below audibility and registers at the minimum audible level in five decibel steps. Our zero line therefore is placed below any possibility of being in hearing range. Our reports, however, will use accepted standards.

The hearing acuity of children in the five to 15 year age group seems to be slightly less than that of the adult. This is most noticeable in the lower tone range. Children's responses vary, but we believe their alertness and interest during the first tests, more than neutralizes the claimed gains in performance presumed to be due to the familiarization caused by repeated testing. Figure 1 shows the averaged testing of 40 ears in which the first test is compared with subsequent audiograms. This chart shows the hearing response obtained was less acute on additional examinations.

Our audiometricians, Miss Bollback and Miss Brewster, are of the college graduate level and they were trained by us. They are reliable. They have been of great service in compiling our statistics. Mr. James McKernon Jones had his technical training under our direction. He is a graduate in business administration, which training aided him in organizing and supervising our technical work.

Our audiometers are the usual stock variety. They are constantly tested for accuracy in our own laboratories.

All the surgery done in connection with our project was marked "special." All such cases were carefully handled by a selected staff, the co-authors of this paper.

Our dietary program was under complete control of the authorities at Mount Loretta Home, Staten Island, New York.

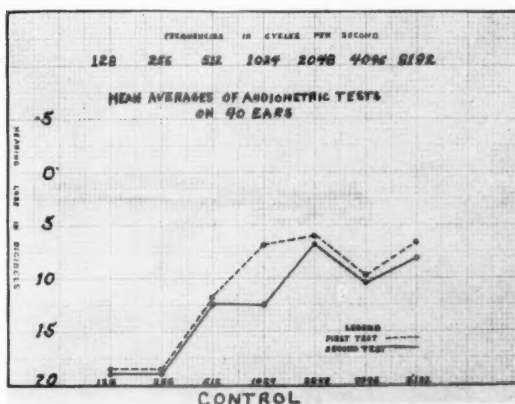


Fig. 1.

Every patient with impaired hearing has been personally examined and classified by one of us (Jones). Private patients of the writers have been included in this report.

We have studied the result of tonsillectomy and adenoidectomy on normal hearing patients. One hundred such normal hearing patients were selected from our "Tonsil Clinic."

The criteria for selection of normal hearing children were the following: We chose children between the ages of five through 16 inclusively whose hearing loss was no more than 20 db in the "key" frequencies (i.e. 512, 1024, 2048, 8192); whose past history and clinical examination suggested that their ears were free from pathological changes caused by infection; whose cultural backgrounds were similar. An attempt was made to select children who showed the same average intelligence.

The results of the tests on these 200 ears are of sufficient interest to report (Fig. 2). This chart shows the averaged improvement following adenoidectomy for all tones except the 256 frequency. The greatest improvement was for the 2048 frequency. Guild's³ observations are interesting in this connection. He stated, "The average change in hearing acuity for low tones that occurred in children is so small that it would not be considered significant in

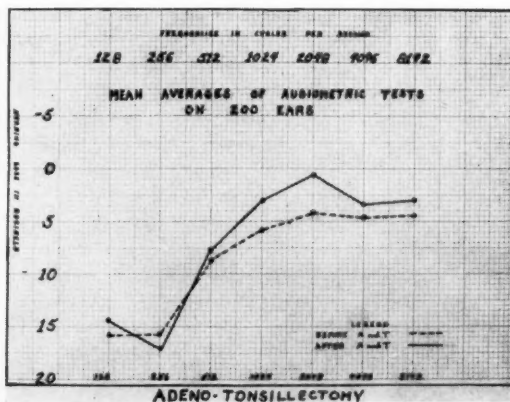


Fig. 2.

comparing tests of the individual, but it occurred so consistently in the groupings of the material of the present study that I believe it to be a fundamental phenomenon in the age changes related to hearing: certainly it should be looked for by others who have material suitable for the purpose." Our observations regarding improvements in hearing with increasing age agree with Guild's expressed belief. Our opinion differs from his however regarding the relationship between the adenoid tissue and hearing impairment. It is generally accepted that the adenoid tissue, under normal circumstances, atrophies with age. There is concurrent improvement in hearing as stated by Guild. Since the removal of tonsils and adenoids apparently produces an immediate similar improvement in hearing, we believe that the suggested normal increase in hearing accompanying aging is also due to adenoid regression or elimination. Irradiation of lymphoid tissue in the nasopharynx may act in a similar manner with like results.

The next group we wish to report on was composed of children who had impaired hearing previous to operation. Study of this group continues and at present we will report only those patients in whom hearing improvement has occurred. In no instances was removal of tonsils and adenoids done with the intent of improving hearing. In each instance the removal was done for other adequate

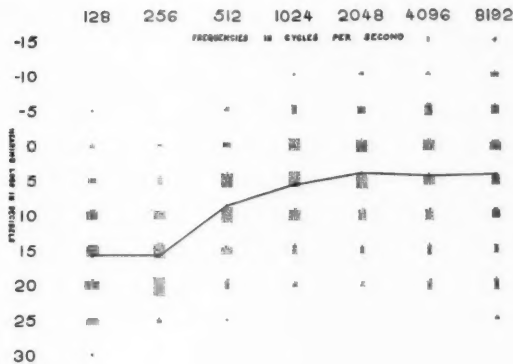


Fig. 3.—Before tonsillectomy and adenoidectomy. Distribution of the cases tested.

causes (Figs. 3 and 4). In connection with our observations as to the effect of adenoidectomy we will interpolate a theory regarding recurrence of adenoids after acceptable surgical removal. Our observations are that adenoid tissue may recur if irritating or infecting factors remain after their removal. Such factors are sinusitis, allergies or both. We do not believe that the adenoids recur frequently following technically adequate surgical removal in the absence of such factors. The hypertrophy of lymphoid tissue which commonly occurs between the posterior tonsillar pillar and the posterior pharyngeal wall can be caused by the post-nasal drainage from chronic sinusitis. The hypertrophy of lymphoid tissue around and in the eustachian orifice could be caused by the same process.

The next, or third, group which we studied has become united with what was originally to have been our fourth. Sinus inflammation and allergy have been treated as a single problem. We are safe in claiming an intimate relationship. Fowler⁴ claimed that of 100 children with impaired hearing whom he studied, 57 per cent had moderately severe involvement of the nasal sinuses and 86 per cent showed at least some positive pathologic changes in the sinuses. He also stated⁵ that treatment of the sinusitis produced marked improvement of hearing in 25 per cent of the children and definite improvement in 50 per cent. We can state that repeated attacks of sinusitis

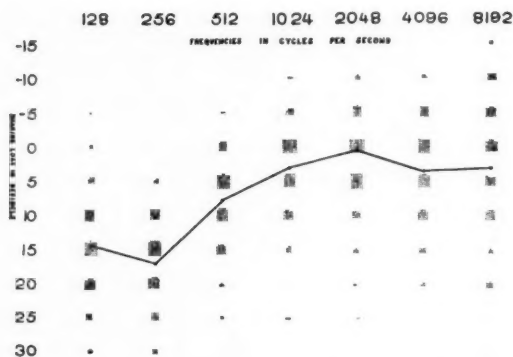


Fig. 4.—After tonsillectomy and adenoidectomy. Distribution of the cases tested.

do cause a hearing loss in children and that treatment of the sinuses will restore that loss to a varying degree. We have observed a permanent restoration of hearing to within normal limits by eliminating a common underlying cause of recurrent sinusitis, allergy. Our claim that allergy can cause hearing impairment is supported by other observers. Hansel⁶ stated, "Deafness in children may be the result of eustachian tube involvement or serous otitis of allergic origin." Rinkel⁷ stated, "Finally there is the classical type of perennial nasal allergy characterized by more or less constant obstruction of the nares, copious discharge of mucous from the nose and the nasopharynx. In winter time it is the rule that the discharge is semi-purulent and often is wholly purulent in appearance. Sneezing is a prominent symptom, sleep is often impossible without nasal medication. Children with this type of syndrome often become mouth breathers. They often have impaired hearing." Jordan,⁸ stated, "Allergy plays an important role, either as a cause or a contributing factor in deafness." Dean,⁹ in 1937, Lewis,¹⁰ in 1929, Proetz,¹¹ in 1931, Lillie,¹² in 1944, and Ashley,¹³ in 1949 presented evidence as to the importance of allergies as a cause of impaired hearing. There are many references to the association of allergy with Ménière's syndrome. Williams¹⁴ stated, "The cardinal symptom of Ménière's disease is usually stated to be vertigo. The cardinal sign, however, appears to be inner ear deafness involving the low tones

rather than the high, and more apparent on the bone conduction curve determined with the audiometer than the air conduction curve." It would seem natural to expect a cochlear disturbance from any cause which would produce vasomotor changes in the vestibular organ. Day¹⁵ in 1950 stated, "More and more evidence is accumulating to support the theory that an autonomic dysfunction or intrinsic allergy is the basic cause of this disorder (Ménière's Syndrome) which might be called vaso-motor labyrinthitis." He also stated and furnished evidence to support the statement that the cochlear part of the labyrinth is the first affected by allergy.

Perhaps we should here state, that which most of us admit to ourselves, that we do not know too much about the causes of impaired hearing in adults. We do know however, that in most instances the non-suppurative chronic hearing impairment takes place gradually. It frequently starts undetected during childhood. Otosclerosis has rarely been found either clinically or pathologically earlier than the age of puberty. Yet, since this disease is commonly of the slowly developing type and since it frequently becomes apparent about the age of puberty, it surely must have had its beginning at some stage during childhood. Observers have commented on the vascular and vaso-motor system as being involved in the otosclerotic process. Wolf¹⁶ stated, "I predict that when a complete reconstruction of the detailed terminal blood supply of the adult petrosa is made, one will find the areas of more or less terminal capillaries with little or no chance for anastomosis to be the sites of pre-deletion for otosclerosis." What could be more logical to suppose than that such a process might occur during the formative stage for the temporal bone and at a time of life when allergic disturbances persistently affect the structure in question. Hilger,¹⁷ also has focused attention on the vaso-motor channels of the inner ear as frequently being affected by the allergies.

Another phenomenon due to vaso-motor disturbances which is quoted by Williams¹⁸ as being associated with allergies, is sludging of the blood. This clumping of cells, bound by plasma, has also been considered in its association with otosclerosis. Our investigations are also concerned with the connection between the typical degenerative processes found in pathological otosclerosis and the allergic phenomena.

At this point the perennial question should arise, "What is allergy?" It is a question we shall leave unanswered. We do know that the structures of the body involved in the term allergy are in an almost unknown and little understood category. The autonomic nervous system with its vasomotor controls, the endocrine system which has recently vied with antibiotics for a place on the front page of our newspapers, and disturbed emotions are assuming increasing prominence in the research field.

We believe that such items as we have mentioned plus many we have not named are inextricably intermingled and they produce an increasing variety of symptoms known as allergic. Allergic symptoms can be caused and in most instances are caused by human environment. Fear will cause a blanching of the face, sensations of heat and cold, sweating, diarrhea, nausea, aphonia, blindness, dizziness and deafness. These may be the symptoms of an acute allergic episode. Sunlight, temperature changes, humidity changes, air conditioning, draughts, and barometric changes can cause sneezing, stuffy nose, palor of the nasal membranes, vascular headaches, and diminished hearing. Again we cite allergic symptoms. We all recognize pollen, dust, animal dander, plus many other inhalants, as causes of allergy. Bacteria as causes of allergies have their supporters. We wish to stress the ingestants as being important, perhaps the most important causes of allergies which affect the ears, nose and throat. Ingestants such as milk, eggs, wheat shellfish, chocolate and nuts have been proven the principal offenders in our experience.

Our monumental job is to begin classifying the causes of impaired hearing and treat the probable causes. Allergies would appear to be one of the causes to include in such a classification. We have published our method of anti-allergic treatment under the title, "Allergy—Effective Treatments Used In Otolaryngology."¹⁹ This treatment is essentially the elimination of contacts, desensitization with the minimal dose methods of Hansel,²⁰ and aid in the physical and mental rehabilitation of the patient. We prescribe a selection of foods which have minimal allergin content with maximum protein and complete nutritional values.

Scientific absolute proof is difficult to obtain and empirical medical claims are seldom of a nature to be entirely convincing. Deductions from circumstantial evidence must be relied upon in most instances however to effect medical advances.

COMMENT

We have presented the information we have obtained to date. As the work progresses, we hope to bolster our claims by impressive numbers. Thus far our data are based on painstaking individual observations.

Our main objective is to improve, cure, or prevent impairment of hearing in children. We have an increasing hope that as a final result of our studies we can furnish convincing proof of the relationship between allergy and non-suppurative impairment of hearing.

120 E. 60TH STREET.

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DISCUSSION

DR. J. M. SUTHERLAND (Detroit): I have nothing particularly to add to Dr. Jones' paper. I enjoyed it very much. I should like to have Dr. Jones describe his particular method of doing his testing and also how much stress he lays upon nutrition in treating these children.

I have had several of these children with more or less impairment of hearing associated with stuffy noses—with or without nasal secretions—under my observation and treatment since I begin using anti-allergic methods some four years ago. I formerly referred my allergic patients to an allergist. This was unsatisfactory, because you lose contact with your patient.

After I became more deeply interested in allergy, I discontinued the scratch test, and now use the intradermal testing method for all foods, inhalants, pollens, etc. I do not know yet just how reliable many of these tests are—especially for foods. Many children show very definitely positive reactions to different foods when tested intradermally. Most of these foods are eliminated from their diets for various periods of time.

The $\frac{1}{4}$ cc tuberculin syringe and a 27 gauge $\frac{3}{8}$ inch intradermally pointed needle are used exclusively in testing. I use a different syringe and needle for each allergen. Each syringe and needle are kept in a separate small bottle. Each allergen number corresponds to the number of the syringe and the number of the bottle in which the syringe is kept. This method positively prevents contamination of your allergens.

The lumen of the barrel of the $\frac{1}{4}$ cc syringe is so small that the amount of allergen can be better controlled. The 27 gauge needle is so small that it can be introduced with little or no pain and with the minimum amount of trauma. Seldom more than .01 cc is necessary for testing children, and never more than .02 cc of any allergen is injected. Up to this time, I have never experienced an unfavorable reaction. This method can be used on small children; usually a fancy looking all day sucker does the trick and the child lays down on the lounge and the game begins. I have found that many of these children are quite sensitive to inhalants such as dust, molds and epidermals, as well as to pollens. In such cases, a hypodermis sensitization allergenic extract is given as part of the anti-allergic management.

Some of the most startling results have occurred in children where a secondary tonsillectomy and adenoidectomy failed to relieve their nasal symptoms—especially

a few whose nasal cavities were filled with a muco-purulent secretion which had to be removed with suction.

In these particular cases, elimination diets, supplemented with intravenous vitamin therapy biweekly for three or four weeks, and desensitization treatments seems to offer the best results. The hearing improved to normal in most of these children. The most gratifying improvement was in the child's breathing.

It is a pleasure to listen to such a practical paper, and I wish to thank Dr. Jones for presenting it before our society.

DR. CHARLES E. KINNEY: Mr. President, Members and Guests; hearing in childhood has been for a long time one of my major interests. I wish to compliment Dr. Jones with particular reference to the slides marked "A and T" rather than "T and A," first stressed by Meltzer and others. This puts the adenoid part of that surgery in its proper place.

I would also like to mention that although I do not find as many cases of this phenomenon as Dr. Jones is finding, I have found some that I could put in that classification. I have observed distinct changes in the appearance of the tympanic membrane as you watch the progress of these children towards what I call normal.

We are now starting to get some kodachrome pictures of these tympanic membranes to see if we can actually confirm by the picture our mental impression, particularly the change in the color, as pointed out by Dr. Hoople. I would like to ask Dr. Jones if he has observed changes in the appearance of the tympanic membrane that go along with these audiometric improvements. It is a very practical field and I compliment the essayist.

DR. EDMUND P. FOWLER, SR.: There is no question but that allergic conditions may affect the hearing but it is surprising how many people have severe allergic attacks without any effect upon the hearing.

In the early days we did not know quite as much about allergy as we do know. We did not have the antihistaminic drugs and we did not have the various tests but we knew the child had a stuffy nose and we knew that the ears were inflated with more difficulty. Unless the swelling does affect the middle ear then the child does not seem to have any coincidental change in the hearing.

I also have kept for many, many years (25 years or so), charts of the children showing the changes in the hearing. Also the x-ray findings. Every child had repeated audiometer tests and the surprising thing was that the variations could not always be correlated with the allergy. Not only this but in several hundred school children in the New York schools examined at a year interval we found changes quite comparable to those shown on the slide here shown. But my children had received no treatment whatsoever. It was remarkable, that even a 60 db loss in some of them, returned to a normal hearing level. It may have been their allergy that did this because allergic lesions change without any treatment. It is very difficult to correlate definitely what we have done with the results that we see. I would like it if Dr. Jones would—and I am all in sympathy with this—say something about the subject mentioned in the title of the paper. The methods that were used, because that is the important item.

Thank you!

DR. HENRY L. WILLIAMS: I have enjoyed this paper of Dr. Jones very much indeed because I think that he is stressing a very important point. However, I would think that he might give attention to some other measures directed towards the alteration of that threshold above which allergic manifestations occur other than the direct antigen-antibody type of therapy, such as, that which is secured by hyposensitization, or even by elimination of antigens in the diet, or contactants.

I think you are all quite well aware of the fact that some people will react allergically to a food stuff at one time, which they can take with impunity at another. So certainly the state of the individual at the time he comes in contact with the allergin is of great importance.

There are several things which influence this allergic state. One of the most important things in our experience—I think this was brought out by Dr. Lillie some years ago—is the effect of the unsaturated fatty acids upon the allergic state of the individual. It has been shown particularly by Arild Hansen that the lack of an adequate supply unsaturated fatty acids in the diet tends to lower the threshold above which allergy occurs.

Trace minerals, which have been featured by Dr. Sam Roberts, and which I sometimes make jokes about, I really believe to be of tremendous importance especially in children. A great many children don't get adequate supplies of these minerals, particularly in sections of the country where the soil has been depleted of these minerals by long cultivation.

The question of vitamins might also be brought up. It is assumed that most children and adults secure adequate vitamin supply through diet. This is not always so because certain British investigators have shown intestinal bacteria may have a tendency to destroy vitamins in the bowel, so a person may develop an avitaminosis on an apparently adequate vitamin intake. So occasionally in certain individuals giving such substances intravenously might be indicated.

I think attention to the general condition of the patient is of at least equal value in many of these individuals to that of so-called specific allergic treatment.

DR. SUBRAMANIAN: I enjoyed Dr. Jones' paper. I just want to ask three questions for my own benefit. One is, what is the routine line of treatment that he gave all these children and what is the duration of treatment, and whether the effect of the treatment was tested after a year to see whether the improvement was permanent or was only temporary?

The other question is, since most of his work has been done on children, I would like to know what he thinks about its effect, whether it is on the nose and the nasopharynx, or whether he considers it to be in the cochlea and the cochlear depression?

The other question I would like to ask is, whether they were all routinely examined by the nasopharyngoscope before and after treatment?

DR. ROBERT C. McNAUGHT: I too enjoyed Dr. Jones' paper very much. I was particularly impressed by a provocative statement that allergy might have an etiological relation to clinical otosclerosis, if I did not misinterpret the remarks that have to do with the vascular component of allergy. The implication was that vascular changes accompany allergy and may cause otosclerosis. I think most people interested in otosclerosis have probably noted as I have, a much higher

incidence of hayfever and other manifest allergy in otosclerotic patients than in the public at large.

I had always considered that that was a genetic coincidence rather than a cause and effect relationship; that the same constitutional inheritance that predisposed to allergy was linked in some way with other factors that lead to otosclerosis. This thought of Dr. Jones, though, is worth considering.

PRESIDENT DAY: Is there any further discussion? Dr. Lurie!

DR. MOSES H. LURIE: Mr. Chairman and Members, I would like to ask Dr. Jones whether he noticed an increase of allergy in children during the past ten years. In my experience in private practice there are more children coming to me today with allergy and deafness as the result of blocking of the eustachian tube than there were ten to fifteen years ago. I have a feeling it is due to the use of antibiotics and as a result we are developing new sensitivities in our youngsters to the low grade organisms that are present in the nasopharynx and in the nose. These youngsters I find are extremely hard to treat. They show no evidence of any food allergy and they all show more or less some reaction to some of the non-virulent bacteria of the nasopharynx. I also found that a great many of these children have been treated with the excessive use of shrinking solutions in the nose and then developed intense allergic reactions to the shrinking solutions. I think one of the greatest mistakes that we as otologists make is the excessive use of these shrinking solutions in the nose and nasopharynx in an endeavor to cure the acute colds.

There is one condition that I would like to ask Dr. Jones, if he has run into it because I encountered it about three or four times and I think it is more prevalent than most of us realize. It is a condition that develops in young children which is known as chronic fibrosis of the pancreas. These children all show marked mucous changes of the nose and nasopharynx and even of the middle ear. As I said, this condition has come to my attention only in the last few years. It is the fact that the pediatricians have just discovered it and are now able to do tests on this condition in the past five years.

PRESIDENT DAY: Is there any further discussion? If not, I will ask Dr. Jones to close.

DR. MARVIN F. JONES: When allergy ceases to be a controversial subject, interest will also drop.

Dr. Sutherland brought out the methods of testing the nutritional value in the private practice experience in the testing for foods.

It will take quite some time, or would take some time to cover all of the discussions, but I will cover as many of them as I can. I do not use skin tests any more for foods. I found skin tests for food to be misleading. You can get positive skin tests with a negative clinical result and you can get a negative clinical test with a positive skin test. So I have discontinued skin testing for foods entirely. I now use a selection diet, in other words, selecting foods that are poorest in allergen content, as I stated in the paper.

There are two skin tests that however I do use. One for histamine (method of Hansel) and I also use the skin test for dust.

I first started work on allergy with an allergist. About 1920 I believe it was.

My work has largely been on the nutritional type. The oldest case that I have on record has maintained normal hearing for 19 years. He has just gone in the Army. Most of the cases I have reported today are from private practice, not cases that occurred in the clinic. Cases where hearing has improved following diet treatment are not common but they do occur and that is the point that I want to make.

We are struggling to find out all the causes of deafness in children or impaired hearing in children and the proposition that allergy and proper anti-allergic treatment can produce improvement in hearing impairment is certainly worth substantiation.

I made the statement when I first started the paper that the title of this paper was poorly phrased, "anti-allergic methods used as a treatment in the restoration of hard of hearing."

The nutritional value has been brought up by several discussers. I have not discussed the nutritional phase of diet today but it has been thoroughly worked out and the suggested diet is in nutritional value. Two criticisms have been the lack of calcium in the diet, from eliminating milk, and the lack of vitamin B complex. The vitamin B complex is deficient in practically every urban diet. We don't feel too concerned about it but occasionally we supplement vitamin concentrates for our patients. For the benefit of those who listen to the milk propaganda people we occasionally put some calcium in the diet as a supplement. I think there is an adequate amount of calcium in the prescribed diet.

Also I might take up the use of the nasopharyngoscope. The use of the nasopharyngoscope has, within recent years, become a routine procedure. Previous to that time and previous to the work at Hopkins, it had not been used routinely on cases. Some of those cases are today included here.

As regards the occurrence of allergy and not having hearing loss associated with it, I refer Dr. Fowler to the paper that was given here yesterday morning, in that you can have a traumatic condition, vascular insult to the inner ear and that can cause impairment months after the actual insult occurs, so unless you follow the cases over the years and watch the diet and see what effect there was it would be impossible to say there is no connection. Although impaired hearing does not occur immediately, it can occur later. I believe Hilger's work bears out the fact allergy can not only affect the inner ear and cause deafness but it can affect the middle ear and cause a middle ear type of deafness.

As to treatment, I will say for Dr. Subramanian, the treatment is very thoroughly described in a paper which I presented before the Triological last year in San Francisco, and I will be glad to give that to Dr. Subramanian. It answers his question on treatment, including the duration. It is a very tedious process. Occasionally improvement is sudden. The permanency is answered in the earlier discussion in that these cases that I presented, probably ten out of the twelve of the records I have shown, held their improvement over the five-year period. I think there are only two or three slides that were taken from our more recent investigations in the conservation of hearing clinic.

Dr. McNaught's surmising query on clinical otosclerosis is absolutely right. Dr. Wolf has mentioned the possible connection between allergy and otosclerosis, as has also Dr. Hilger. Regarding the increase incidence of otosclerosis during the past years, in children I believe that the emotional factor has not been properly estimated in its relation to allergy. I believe the incidence is increasing and cer-

tainly children are more emotionally upset today than they were during past years, as are also the adults.

PRESIDENT DAY: The last paper on the morning's program is "The Effect of Cholesteatosis on Bone" by Dr. Theodore E. Walsh.

PRESIDENT DAY: This paper is now open for discussion.

Dr. Williams!

XCVIII

THE EFFECT OF CHOLESTEATOSIS ON BONE

THEO. E. WALSH, M.D.

WALTER P. COVELL, M.D. (By Invitation)

AND

JOSEPH H. OGURA, M.D. (By Invitation)

ST. LOUIS, MO.

The purpose of this paper is to examine the evidence, clinical, histological and experimental to try to determine the effect on bone of cholesteatosis. The term, cholesteatosis, borrowed from our English colleagues, is used in preference to cholesteatoma because the latter term indicates a neoplasm. Although primary cholesteatoma, the result of misplaced embryonic epithelial rests, does occur in the cranial cavity, it is a rare condition. Primary cholesteatoma is even more rare in the temporal bone and should not be confused with cholesteatosis which is the result of inflammation and altered aural physiology.

The destructive effect of cholesteatosis on bone has long been recognized. Many considered the effect to be one of pressure due to accumulation of desquamated epithelial cells which were unable to escape through a narrow communication with the external ear. Others felt that there was some definite chemical process responsible for the lytic effect on the bone. Cholesterol was thought to play a part in this lytic process. Still others supposed that the connective tissue matrix had an effect on the underlying bone and was responsible for its destruction. It is important to determine what factors are really responsible for bone destruction so that surgical treatment may be based on sound premises.

There are many cases which seem to lend credibility to the theory of pressure necrosis. It is not uncommon to see patients with a small attic perforation through which a small amount of discharge

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and the white flakes of desquamating epithelium may be obtained. At operation, these cases are often found to have in the antrum and epitympanic spaces, and indeed throughout the mastoid, a large epithelial cyst full of the amorphous material characteristic of cholesteatosis. It is not difficult to suppose that as the desquamated cells could not escape through the small perforation the effect was that of an expanding tumor with destruction of the underlying bone. There are other cases, however, which make one doubt the plausibility of this theory as the sole cause for bone destruction.

REPORT OF A CASE

E. K., a 12 year old boy, was seen first in the Nose and Throat Clinic in December of 1949. Past history was that he had had a draining left ear since the age of four, the result of measles. He had some kind of operation on the ear when he was four years of age but the ear continued to discharge and two years later he had a radical mastoidectomy. Since then the ear has continued to discharge. On examination in December, 1949, the posterior part of the left radical mastoidectomy cavity was found to be completely epithelialized and clean, but the middle ear and epitympanic regions contained granulations and foul pus. This cavity was treated by cleansing and various local medications. One of us (T.E.W.) saw the boy for the first time in February of this year. On examination at that time, he complained of having occasional dizzy attacks for the past few months and he had a very definitely positive fistula test. At operation, cholesteatosis was found in the antrum and middle ear region and on elevating the subepithelial matrix, a fistula into the horizontal canal was found. It seems impossible that this fistula could have been caused by pressure because, following his first radical mastoidectomy, the cavity and middle ear were completely open to the outside and no accumulation of epithelial debris occurred in the middle ear. Granulations and suppuration were still present, however. Cases similar to this are not infrequent in our clinic.

EXPERIMENT

In order to investigate the possibility that some form of chemical lytic process was responsible for bone destruction in cholesteatosis, extracts of material removed at operation were made. The matrix and desquamated epithelial debris obtained at operation was ground in a mortar with saline. The extract was passed through a Berkefeld filter and tested for sterility. Into this sterile filtrate were immersed ossicles removed from patients on whom fenestration had been performed for otosclerosis. Similar ossicles were immersed as controls in normal saline and were kept in an incubator at 37° C. for varying intervals of time. The ossicles were then sectioned and examined histologically. Table I shows the results. It was found that after incubation of an ossicle in filtrate from material obtained in cases of cholesteatosis, no striking histologic changes were found, how-

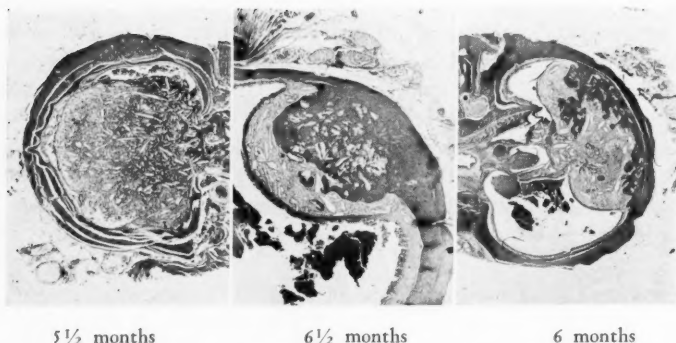


Fig. 1.—Photomicrographs of sections of rabbits' bulla into which cholesterol crystals had been placed.

ever, when the experimental ossicles were compared to controls, there was an indication, though minor, of some slight alteration in the filtrate-treated ossicles. These changes were briefly, that the bones stained poorly and in most instances were very pale and unevenly stained. The calcified cartilaginous rests apparently had undergone some necrosis. There were areas of necrotic bone present in some specimens. These necrotic areas were located rather more deeply than superficially. The changes were not dramatic nor particularly significant but it would seem that further investigation along this line might be worthwhile.

Lautenschlager¹ believed that chemical lysis played a part in bony destruction associated with cholesteatosis. He felt that cholesterol was responsible for such lysis. To investigate this possibility, animal experiments were performed in which cholesterol crystals were implanted in the bulla of eight rabbits. The bulla was opened with some damage to the mucosal lining and crystals were placed in the cavity and the skin closed. Eight animals were used in this experiment and were sacrificed at intervals of from one day to 25 months after the procedure. In four of the eight animals there was new bone formation (Fig. 1), presumably as a reaction to the presence of cholesterol crystals. In the animal killed after one day, only blood was seen in the bulla. In one animal sacrificed 17½ months after the insertion of the cholesterol, the bulla apparently was nor-

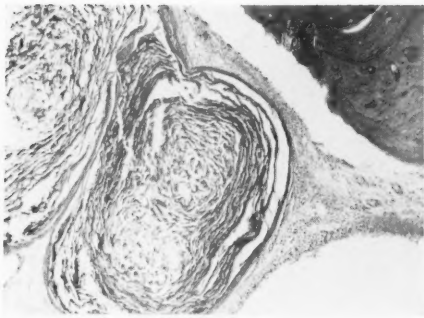


Fig. 2.—Photomicrograph of section of bulla of rabbit into which a homologous skin graft had been placed 24 months. There is no new bone formation seen.

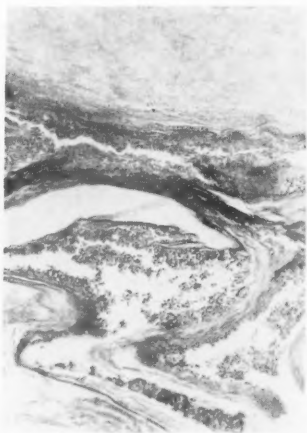


Fig. 3.—Photomicrograph of section of rabbit's bulla into which human material from a case of cholesteatosis had been placed. 4½ months later—remains of epithelium and pus. No bony changes.

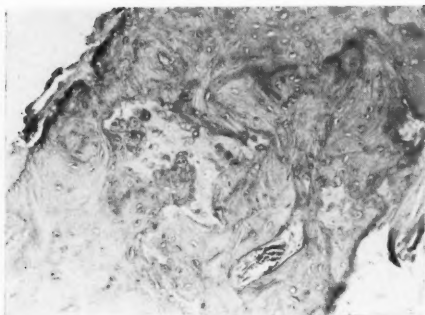


Fig. 4.—Photomicrograph of an area in the body of the incus showing an early stage in the changes within the calcified cartilaginous rests. The calcified rests return to a hyaline state, the nuclei are irregular and variable in size and show a tendency to cluster in small groups.

mal. In no animal was any bone destruction found as a result of the indwelling cholesterol.

Milstein² in experiments on 24 cats and eight dogs, exposed the bulla, removed the mucosa and skin grafted the cavity. He found that while he produced a "take" readily in the cats, it was not always possible in dogs. He concluded from his experiments that active inflammation was necessary for active proliferation of the epithelium in such animals. In order to repeat these experiments, we used rabbits. A split thickness graft measuring approximately 3x3x3 mm taken from the abdominal wall was placed in the bulla of the rabbit opposite the opening. These animals were allowed to survive between four days and 25½ months. The graft took in only one animal (Fig. 2), and here is seen a mass of skin growing with desquamation of the superficial layers of the epithelium. Nine animals were used in this experiment. Although in all of them acute and subacute inflammation was found, there was no evidence in any of bone destruction.

In a further six animals, we implanted material, removed from cases of cholesteatosis, in the bulla of rabbits, but in no case was any change other than acute and chronic inflammation found in these animals. It was not surprising that heterologous material should fail to take (Fig. 3).



Fig. 5.—Photomicrograph of an area through the long process of the incus to which a cholesteatotic process was attached. The bone is undergoing necrosis; the blood vessels are surrounded by invading matrix; and peripherally the cartilaginous rests are undergoing necrosis.



Fig. 6.—Photomicrograph of an area in the head of a malleus to which a cholesteatotic process was attached by its matrix. The matrix has invaded and replaced necrotic areas of the bone. There is some attempt at new bone formation by osteoblasts but the greater part of this ossicle was eroded.



Fig. 7.—Photomicrograph of a horizontal section through the petrous bone showing cholesteatotic mass in place in the tympanic cavity.

HISTOLOGY

The ossicles removed at operation from cases of cholesteatosis (over 100) were routinely examined histologically. The outstanding feature of these ossicles was their irregularity and the evidence of necrosis. The sequence of changes which led to this necrosis seemed to be, first, a return to a cartilaginous state of the calcified cartilaginous rests. Calcium seems to be lost and the nuclei of the cartilaginous rests become clumped and vary considerably in size (Fig. 4). Necrosis results with a final disappearance of the rests. The subepithelial connective tissue (cholesteatotic matrix) invades the bone of the ossicles by direct extension along blood vessels (Fig. 5 and 6) and may extend for a considerable distance beyond the immediate point of its entry. Osteoblastic and osteoclastic activity is in evidence (Fig. 6). In some regions in which there is invasion of the fibrous connective tissue, new bone formation is to be found as well as osteoclastic activity, but the destruction of bone is much more in evidence than the laying down of new bone. There is some periosteal bone proliferation underlying the matrix and it seems likely that this new bone is probably the result of infection and is responsible for the irregularities in the contour of the ossicles.

Various factors seem to be operative in the dissolution and necrosis of the bone. First, there seems to be an osteolytic process

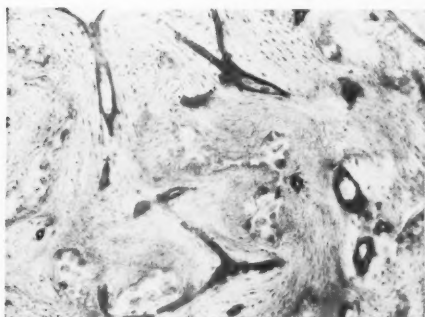


Fig. 8.—Photomicrograph of enchondral layer of the capsule. The calcified cartilaginous rests are prominent but show no degenerative changes in this particular area. The bony channels for blood vessels show a deposit of deeply stained substance.

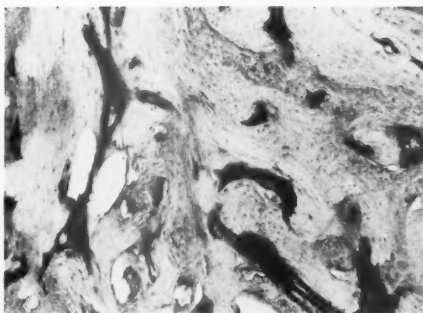


Fig. 9.—Photomicrograph of the enchondral layer of the capsule showing necrosis of bone and disappearance of calcified cartilaginous rests. Areas occupied by blood vessels are represented by the darker stain.

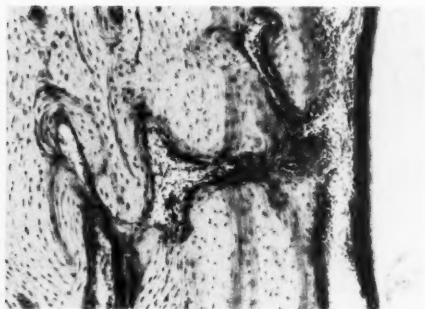


Fig. 10.—Photomicrograph of cholesteatotic matrix invading periosteal layer of capsule by following blood vessels.

occurring in the calcified cartilaginous rests. The dissolution of bone also seems to be accomplished by the invading matrix. The actual process of osteoclasia seems to be stimulated by secondary infection and this may be an important factor. Interruption or interference with the blood supply of the ossicles may result in the deeper necrotic areas in the bone. Periosteal bone proliferation is usually localized and immediately underlies the mucoperiosteum. It is apparently also stimulated by cellular infiltration and edema of the mucous membrane. Replacement of necrotic areas of the bone by connective tissue occurs with final fragmentation and dissolution of the remaining bone. It is the endochondral layer of the bone which is primarily destroyed. The connective tissue matrix penetrates the periosteal layer through the blood vessels and the cartilaginous rests in the enchondral layer become necrotic and disappear.

The temporal bone of a 10½ year old white female who died of lateral sinus thrombosis was examined and cholesteatosis found in the middle ear cavity (Fig. 7). This mass of cholesteatosis equals 5.7 mm by 5 mm by 2 mm. The mucous membrane of the tympanic cavity is edematous and thickened. The following bony changes were found in this specimen: 1) Irregular periosteal bone proliferation. This may be due to the inflammatory process and stimulation of the periosteum to new bone formation. 2) There is a

TABLE I

NO.	NO. DAYS INCUBATED	SPECIMENS	FINDINGS
1	10 days	piece of incus	Stains poorly with areas deeper than others.
2	not incubated (control)	small piece of incus	No change.
3	32 days (control)	incus	Only apparent change is uneven staining of blue.
4	32 days (control)	malleus head contaminated with staph.	Few changes in cartilage (articular).
5	32 days (control)	head of malleus	Shows few, if any, changes.
6	31 days	incus	Necrotic changes in calcified cartilaginous rests and articular cartilage; bone stains poorly.
7	31 days (control)	head of malleus	Few, if any, changes.
8	55 days	incus	Areas of necrosis in which bone and cartilage are involved.
9	55 days (control)	head of malleus	Few, if any, changes.
10	16 days	incus	Few areas of necrosis of the bone, however cartilage and rests show only slight change.
11	16 days (control)	head of malleus	Few, if any, change.
12	90 days	incus	Some degenerating cartilage; bone stains poorly.
13	90 days (control)	head of malleus	No change.

blue staining substance about the blood vessels (Fig. 8 and 9), areas of necrosis of bone, chiefly in the enchondral layers (Fig. 9). This is considerably more pronounced than in most specimens of ossicles removed at the time of surgery. It is possible that some of the vascular changes may be due to septic thrombi in this instance. There is seen a penetration of matrix into the bone about the blood vessels (Fig. 10). At times it is quite far removed from the point at which it originally started to invade. Necrosis of this bone is evident around such penetrating fingers of the matrix.

DISCUSSION

There is little evidence that pressure necrosis is the sole reason for bony destruction in cases of cholesteatosis. Many patients seen in the clinic today have had a previously incomplete radical mastoidectomy, in which epithelium with its underlying connective tissue is found closely adherent to the horizontal canal with evidence of necrosis beneath in the form of a fistula. This fistula, as in the case quoted, must have occurred between the time of the first operation and when the patient was finally seen complaining of dizziness, yet, during this time, there was no obstruction to drainage from the cavity nor any evidence at the secondary operation of an accumulation of exfoliated epidermal cells.

Histologic evidence, as displayed in sections of ossicles removed from cases of cholesteatosis and also in sections of temporal bones in which cholesteatosis is found, demonstrates a projection along blood spaces into the bone of connective tissue which underlies the epithelium. There is some evidence in the sections of the ossicles that the bone is changed before such connective tissue proliferation can occur. Whether this change occurs from infection or from some chemical lytic action is uncertain. It seems evident, however, that experimentally cholesterol has no part in any osteolytic process which may occur with cholesteatosis. It is certainly not clear which factors are the most important in the dissolution of bone underlying the matrix in cases of cholesteatosis. Whether infection, chemical lysis or connective tissue growth with the consequent change in vascularity, or all of them play a part has yet to be determined.

Further investigation is necessary to determine the exact processes responsible for osteolysis. The possibility of enzymatic action suggests itself. Again one wonders in what way this epithelium in

cholesteatosis differs from normal skin, for we have yet to see similar changes occurring in cases in which a Tiersch graft has been used to line a mastoid cavity.

It would seem important, however, from these observations that the matrix underlying the epithelium should be removed if any operative work is to achieve success.

WASHINGTON UNIVERSITY SCHOOL OF MEDICINE.

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DISCUSSION

DR. HENRY L. WILLIAMS: I was very much interested in this paper of Dr. Walsh and I think that these factors that he apparently discovered will explain to many of us the effect of cholesteatomas in burrowing their way. Certainly I have come to the same conclusion as he did although I at one time committed a paper, which I very much regret, in which I advocated the method of preserving the cholesteatoma matrix, suggested originally by Nager, and I followed this technic long enough so that I have learned to regret it.

Recently Dr. J. W. Begley collected some of these cases and wrote a paper on cholesteatoma, in which he quoted Dr. Baron, as finding cholesteatomatous cysts recurring as the result of incomplete removal of cholesteatomas. Dr. Baron's conclusions however were not exactly the same as my own.

PRESIDENT DAY: Is there any further discussion of Dr. Walsh's paper?
Dr. Baron!

DR. SHIRLEY BARON: Mr. President, I probably am a "lone wolf" in this gathering because I am one who does advocate the preservation of the cholesteatoma matrix, especially in those cases which are selected for the modified radical mastoidectomy.

Dr. Williams referred to my having described subepithelial cysts, cholesteatomas in nature. Those have been in cases that had been operated on before and in which complete removal of the matrix had apparently been attempted. Many otologists have acknowledged recurrences of cholesteatomas after having removed what they thought was all of the matrix and admit that removing all the matrix without leaving epithelial cell nests is, at times, difficult.

I do try to remove the matrix in those mastoid cases which require revision, since it is not usually possible to know the extent or nature of the primary pathology or the procedure of the previous surgeon.

Dr. Begley and Dr. Williams, in the *Archives of Otolaryngology*, of February, 1951, reported two cases in which cholesteatomatous cysts developed in the post-

auricular mastoid scars. The previous surgery of the first case was done elsewhere and, apparently, it was not known how complete or incomplete was the attempt to remove the matrix. The description of the primary operation of the second case, done in their own clinic, suggests that, by intent, part of the matrix was left in situ and part was removed. In this paper a microscopic section of a cholesteatomous evagination was shown. Such a microscopic evagination could not be seen even with lenses. Dr. Walsh's slides showed instances of deep penetration of the matrix which certainly would be difficult to see in an attempted removal unless, possibly, one used a dissecting microscope in all cases.

I have been, for eighteen years, preserving the matrix in the mastoids of most cases that have had cholesteatomas. I have seen no reason to regret this. I have seen in none of these cases any evidence suggesting innate biological activity of the matrix per se.

I sincerely believe that, in the hands of the mass of physicians doing otologic surgery, preservation of the cholesteatoma matrix would lead to less radical procedures with better hearing conservation.

Thank you, Mr. President, for the opportunity of discussing Dr. Walsh's fine presentation.

PRESIDENT DAY: If I may make a couple of comments, I attempted to preserve the cholesteatoma matrix on a few occasions and I was sorry. If I had to undergo a radical mastoidectomy, I would hope they would remove all the matrix.

Dr. Anson, would you like to comment?

DR. BARRY J. ANSON: On three fundamental aspects of otological anatomy Dr. Walsh's field of study and ours cover common ground. I am glad, therefore, to add a tripart addendum to his excellent paper. The three aspects are concerned with the observation that the otic capsule and the ossicles attain maturity at a relatively early date—either during fetal life or during the first postnatal year.

First, as if aiding osteoclasia, bone is seemingly removed rapidly by halisteresis; consequently, the presence or absence of osteoblasts is not a satisfactory measure of histological activity in the morphogenesis of the capsule and ossicles. Second, the persistence of some of the primordial cartilage in the islands of intrachondral bone often lends the appearance of youthfulness to temporal bones of aged persons. Third, because the developmental steps in the formation of the malleus and incus differ profoundly from those by which the stapes attains maturity in form and adult dimensions, comparative appraisal of pathological change in the ossicles is rendered difficult; for example, while fetal destruction of bone in the stapes is extensive, comparable removal in the malleus and incus is slight.

These features, peculiar to the development of the ear, have been reported at the Society's meetings and have been described in a series of journal articles. We are glad to see them applied to Dr. Walsh's interpretation of changes occasioned by cholesteatoma.

DR. JOHN R. LINDSAY: Dr. Walsh has made a commendable effort to get at some of the basic features in the development of cholesteatoma. He has shown the type of changes that take place beneath the skin, or epidermis, but the question as to the cause of these changes in the bone and cartilage still remains.

There are about three questions to be considered. Is it a matter of pressure? Is it a matter of infection? Or, is the presence of moisture alone the most important feature? If one buries normal skin in a pocket in the bone where it remains moist, cholesteatoma will form.

In an experiment in the monkey, we found that when we laid a tympano-meatal flap over the fenestra in such a way that it was pushed into the fenestra, a cholesteatoma developed that spread right through the labyrinth. If you invaginate normal skin into an area where it is kept moist, the reaction of that skin apparently changes.

The basic question concerns probably the chemical changes which take place in skin when it is placed in an environment of moisture. Many of you have had the chance to watch a cholesteatoma develop in humans over the years. You have seen the development of a pit or cup-shaped hollow in Shrapnell's area, or the posterosuperior quadrant as a result of inflammation. As long as the pit is clean and dry it remains quiescent. If desquamated epithelium or crust collects in the pit and then becomes moist a cholesteatoma rapidly develops.

The skin will not tolerate moisture. Whenever skin is placed in such circumstances that it remains wet, dermatitis develops and rapid desquamation accompanies it. I think perhaps in that reaction lies one of the crucial points as to why a cholesteatoma extends.

Regarding the preservation of matrix, in my opinion there is a reason for removing the matrix which is as follows: It is sometimes impossible to determine by looking at the matrix whether there are small extensions into pits or pockets in the bone beneath the matrix, whether you do not have little pits into the bone into which the matrix extends. If one leaves such pockets there is a possibility of further extensions, particularly in the presence of moisture.

PRESIDENT DAY: Dr. Walsh, will you close the discussion?

DR. THEODORE E. WALSH: I am very grateful to be let off so easily and thank the discussers.

Doctor Baron says his recurrences came in previously operated cases. It is true, of course, that if you revise a case you don't know exactly what has been done previously. I wonder, however, if he watches these cases in which he left the matrix for long enough, he will not find them coming back with fistulae.

Doctor Lindsay brought up the point of moisture. It is true that moisture on the skin will cause an exfoliation and a collection of what we call "cholesteatoma," but the point that worries me is, what is going on underneath. We had a case in point which I would have mentioned in this paper except that I could not find the records. In 1941 when I first went to Saint Louis, we had a woman come to the Clinic with an attic cholesteatoma. She refused surgery. Treating her, one of the house officers bit out the outer attic wall and removed the ossicles. When you looked into the epitympanic space, there was a pit, as Doctor Lindsay described it, about the size of the end of my finger. The patient left town. In 1947 she returned to the Clinic complaining of vertigo. She had a perfectly dry ear. You could look into the cholesteatomatous cavity, which was perfectly epithelialized and dry. She had a positive fistula test. When I operated, I found a fenestra made by nature down to the blue line. It had not gone quite through.

Doctor Anson mentioned the stapes. I wonder if he will tell us if the incus and the malleus show the same variations as does the stapes.

DR. ANSON: No.

I hope I did not give the impression I know the answers. I think there is a great deal of work to be done. I am only suggesting that the investigation might be along chemical lines. There seems to be some evidence, at least, of a lytic process. Perhaps enzymatic action may have something to do with it.

XCIX

EXTRA-TEMPORAL REPAIR OF THE FACIAL NERVE

CASE REPORTS

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ANN ARBOR, MICH.

One of the most gratifying experiences a surgeon can have is to watch the return of function of the facial muscles after a successful operation to repair a damaged facial nerve. Although the results of such a procedure are not perfect, in that varying degrees of mass motion obtain, and fine emotional responses are not restored, they do serve to lift the burden of the grotesque half mask which the patient suffering from facial paralysis must bear. In many instances, the results are magnificent. The excellence of the outcome depends on many factors including the time elapsed after injury, the sagacity and dexterity of the surgeon, the site and type of injury, and the cooperation of the patient.

The literature on facial nerve surgery has been voluminous, particularly since the report of Ballance and Duel in 1932.¹ The vast majority of case reports thus far have dealt with intra-temporal repair of facial nerves injured during mastoidectomy and with decompression operations to relieve Bell's palsy. Many reports of primary suture of the severed facial nerve and its branches after facial laceration are available, but relatively few describing nerve transplant in the soft tissues of the face are catalogued.

Peripheral nerve surgery gained its great impetus from the experimental and clinical work of German surgeons at the beginning of World War I. During this period, Huber^{2, 3} in this country reported on comprehensive experimental studies in peripheral nerve repair, describing the results of auto, homo, and hetero-nerve transplants by many and varied techniques.

Near the turn of the century, substitution anastomosis came to be the accepted method of treatment for facial paralysis. From 1908, sporadic reports of attempts at intra-temporal repair of injured

facial nerves began to appear. In 1927, Bunnell⁴ reported on "Suture of the Facial Nerve Within the Temporal Bone with a Report of the First Successful Case." This operation, performed in 1925, consisted of end to end suture after rerouting of the nerve to obliterate a 12 mm defect.

Bunnell,⁵ in a comprehensive treatise on "Surgical Repair of the Facial Nerve," described suturing of the main trunk of the facial nerve and its small branches after their severance in facial lacerations. Bunnell,⁵ also reported "the first successful intra-temporal repair of the facial nerve done by nerve graft," a case in which a malignant tumor involving the parotid gland and facial nerve had been removed. Repair was accomplished by means of two free grafts from the sural nerve each two and one half inches long, one of which was split at one end to permit anastomosis to three distal branches. Later recurrence of the tumor demanded removal of the entire block of tissue. Bunnell⁵ also reported a case in which the main trunk of the facial nerve was sutured to the three main branches of the pes anserinus to accomplish repair after severance of the nerve by facial lacerations.

Duel,⁶ in 1933, reporting on a series of cases of facial nerve grafts, made brief mention of "one in which removal of the parotid gland had caused a gap, the repair of which required four measured grafts aggregating 150 mm." The end result was not described.

Seeley,⁷ in 1947, reported a case in which a 30 mm segment of the inferior branch of the cervico-facial nerve was used successfully to bridge a traumatic dehiscence in the zygomatic branch of the facial nerve.

Lathrop,⁸ states that he has used successfully a free graft to bridge a gap in the zygomatic branch of the facial nerve which had been sacrificed during the removal of a malignant parotid tumor.

Cardwell,⁹ reported a case in which a parotid tumor was removed and the facial nerve sacrificed. Three 6 cm grafts were sutured to the proximal stump of the facial nerve and their free ends were implanted in the facial muscles. A good functional result was reported.

The four cases chosen for presentation at this time obviously were not selected to demonstrate maximal return of function after

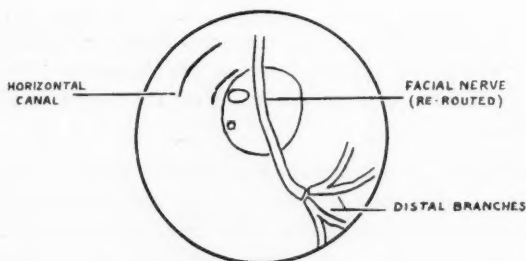


Fig. 1, Case 1.—Diagram showing re-routing of facial nerve and end to end anastomosis at pes anserinus.

nerve repair. One of them, in fact, has a scarcely mediocre result and none is excellent. Rather, they were selected because each demonstrated some unusual features and each presented some particular problem.

REPORT OF CASES

CASE 1.—R. M., white male, aged 14. Admitted to University Hospital, December 4, 1943.

Injuries were sustained on November 16, 1943 when the rear wheel of a truck ran over the child's head after he had fallen from the vehicle. He was unconscious for an unknown period of time and was taken to a hospital near his home in shock.

At the time of his admission to University Hospital (Fig. 2), there was marked asymmetry and deformity of the face due to a fracture of the mandible and multiple fractures of the facial bones on the right side. There was clinical and radiographic evidence of a suppurative process in the right antrum and ethmoids. Anesthesia in the distribution of the second division of the right trigeminal nerve, and complete peripheral facial paralysis on the right were present. Profuse purulent discharge was seen in the stenosed external canal. There was marked hearing impairment of a conductive type on the affected side.

Under conservative treatment, the evidence of suppuration in the ear and sinuses subsided. Testing under general anesthesia demonstrated no response of the right facial nerve to faradic stimulation



Fig. 2, Case 1.—Pre-operative photograph, upper left. Others are photographs taken 18 months after nerve repair.

On November 6, 1943, the maxillary and mandibular fractures were reduced by oral surgeons. Wire bars were ligated to the upper and lower teeth and rubber traction was used.

On December 30, 1943, a right radical mastoidectomy with exploration of the facial nerve was performed. The mastoid was pneumo-sclerotic in type and demonstrated no evidence of suppuration. The external canal was filled by scar tissue which contained loose bone fragments. After the radical mastoidectomy was completed, the facial nerve was exposed from the geniculate ganglion to the stylo-mastoid foramen in which extent there was no evidence of injury. At the stylo-mastoid foramen, however, many bone fragments were seen and it soon became apparent that the styloid process had been fractured at its base, the tympanic plate shattered, and

the facial nerve interrupted a short distance beyond its exit from the stylo-mastoid foramen. At this point it was deemed advisable to permit the child to have a few days of general supportive treatment before attempting the nerve repair.

On January 7, 1944, the second stage of the operation was done. The parotid gland was exposed through an incision extending downward in front of the ear and around the angle of the mandible. Again the mass of scar tissue in the region of the base of the styloid process was encountered. The nerve had been severed at the first branching of the pes anserinus where it was frayed out and embedded in scar. The distal segment was freshened until apparently healthy nerve was obtained which resulted in two distal branches. The proximal segment was then removed from the fallopian canal and re-routed anteriorly which permitted suture of the proximal stump to the two distal branches without tension (Fig. 1).

Convalescence was uneventful after this procedure. On February 3, 1944, osteotomy of the nasal bones was done for correction of the nasal fracture.

Motion of the facial muscles was noted after a period of six months and improvement continued during the ensuing year (Fig. 2).

CASE 2.—R. D., white male, aged 17 months. Admitted to University Hospital May 23, 1949. Five weeks prior to admission, the child tipped over a case of milk bottles, fell on a broken one and lacerated the left cheek. There was said to have been a severe hemorrhage that was controlled with some difficulty. The wound was repaired and healed promptly but facial paralysis was noted immediately.

Examination demonstrated the scar of a healed laceration starting at the tragus, extending downward and forward and ending in an inverted "Y," slightly anterior to and above the angle of the mandible. There was marked induration under the scar and some swelling of the parotid gland. The left eye was tearing and there was complete left peripheral facial paralysis.

On May 25, 1949, an operation was performed under open drop ether anesthesia. The parotid gland contained a large mass of dense scar tissue in which the branches of the facial nerve were isolated

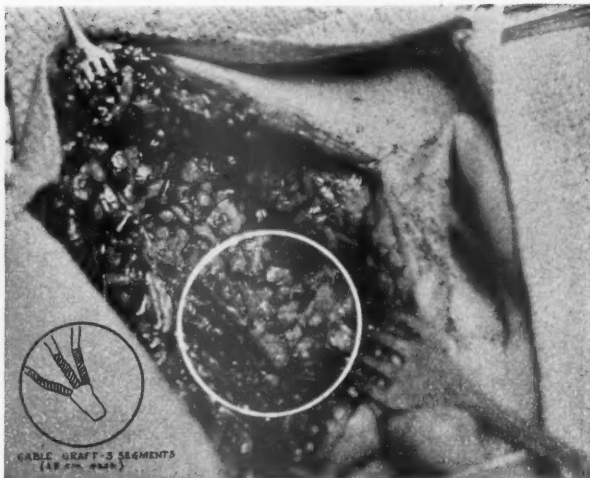


Fig. 3, Case 2.—Photograph of three strand cable graft in place.

Fig. 3a.—Descriptive diagram of Figure 3.

with difficulty. The large branches were found at the anterior border of the mass of scar. These were traced cephalad and terminated in frayed out ends in the scar tissue. The swollen proximal stump was found in the posterior border of the parotid gland embedded in scar. The ends of the nerves were freshened and repair was accomplished by using a cable graft consisting of three sections of the great auricular nerve, each 1.5 cm long. The three ends were sutured to the proximal stump and the other ends of the grafts were sutured to the three distal branches (Fig. 3 and 3a). The post-operative course was uneventful with prompt healing of the wound.

First motion of the facial muscles was noted by the parents at the end of five months. Examination on December 5, 1949, slightly less than six and one half months after operation, demonstrated the patient's ability to close the eye and retract the corner of the mouth while crying.

On March 16, 1951 (Fig. 4) it was noted that the child could close the eye completely and had fairly good motion about the mouth.

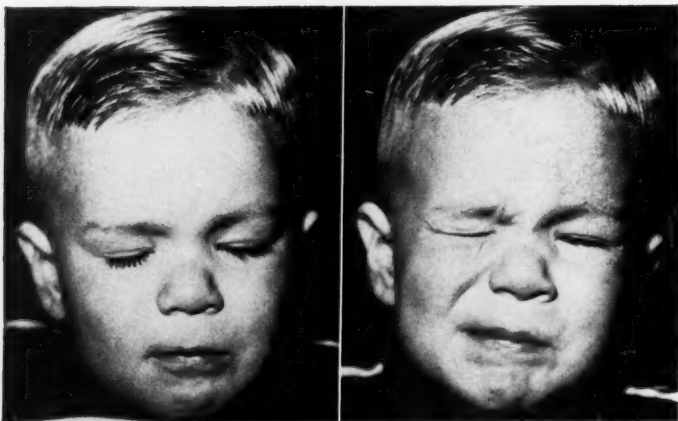


Fig. 4, Case 2.—Photographs taken 1 year and 10 months after nerve repair.

There was no return of function of the frontalis, and the left palpebral aperture was smaller than the right.

CASE 3.—A. C., white male, aged 20. This was a case of facial paralysis due to a bullet wound inflicted on January 13, 1947. The point of entry was at the left zygomatic arch and the point of exit at the upper border of the scapula on the same side.

When this patient was examined, there was a complete left peripheral facial paralysis and a chronic suppurative otitis media on the same side with atresia of the external auditory canal. There was also a high degree of exophthalmos and inflammation of the eye. An internal carotid ligation had been performed in an attempt to relieve the pulsating exophthalmos produced by an arterio-venous aneurysm.

The operation, which was performed on January 6, 1948, consisted of resection of the parotid gland, which was largely scar tissue, a radical mastoidectomy, a Richard's type labyrinthectomy, and the insertion of a 47 mm graft of the great auricular nerve to repair the defect in the facial nerve. It was found that the facial nerve peripherally had been severed just posterior to the pes anserinus. The proximal stump was buried in a mass of scar tissue which also con-

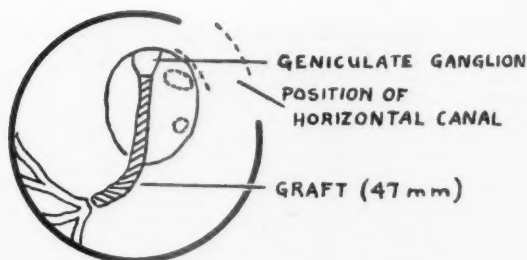


Fig. 5, Case 3.—Diagram showing the site of the 47-mm free graft.

tained the styloid process which had been fractured at its base and dislocated.

In performing the radical mastoidectomy it was noted that the vaginal process was comminuted, and several bone fragments were embedded in the middle ear and in the scar tissue occluding the external auditory canal. The promontory had been shattered exposing the first two whorls of the cochlea. The bone around the oval window was a sequester. A bone fragment apparently had severed the facial nerve in its intra-tympanic course and there was a large neuroma just distal to the geniculate ganglion. The neuroma was excised and the vertical portion of the facial nerve, which was intact, was discarded. The 47 mm segment of the contralateral great auricular nerve (the homolateral great auricular nerve had been severed in the previous carotid ligation) was sutured proximally at the edge of the geniculate ganglion and distally to the main trunk of the facial nerve just proximal to its branching at the pes anserinus (Fig. 5).

The extensive comminution of the tympanic plate and base of the styloid process, and the dense scar tissue in the region of the jugular bulb made it seem likely that the arteriovenous aneurysm had been formed in this vicinity. Fortunately, no particular difficulty was encountered in respect to this lesion, and the further seeking of trouble through exploration seemed unwarranted.

The patient has not been seen by me since his transfer to a distant hospital. He has informed me, however, that the lids were sutured together in March, 1948. The first movement around the



Fig. 6, Case 3.—Photograph taken 3 years after nerve repair.

angle of the mouth was noted in early September, 1948, or eight months after operation. The patient is now working and from a recent communication, seems to be in good health. A photograph (Fig. 6) taken in March of this year indicates that there is still some proptosis. There is said to be some movement in the eyelids although it is not apparent in the photograph. Obviously, the function of the facial muscles is not satisfactory, but the muscle tone appears to be good.

CASE 4.—W. S., white male, aged 27. Date of admission to University Hospital, August 8, 1946.

This patient had noticed a small hard slightly tender oval mass at the angle of the jaw on the left side for about four years. There had been no pain, no facial palsy, and no symptoms referable to the ear.

Exploration and biopsy of this mass had been performed six days prior to admission to the hospital, and had resulted in a complete peripheral facial paralysis. A definite pathological diagnosis was not made from the biopsy specimen.

Examination demonstrated a complete left facial paralysis (Fig. 7). There was a healed incision anterior to the ear with considerable underlying induration. A tumor mass was palpable at the posterior



Fig. 7, Case 4.—Pre-operative photograph.

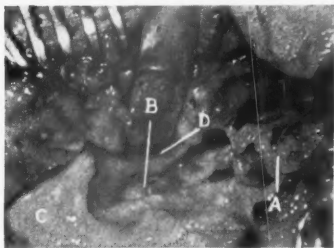


Fig. 8, Case 4.—Photograph taken during operation. A. Tumor elevated from fallopian canal. B. Fallopian canal. C. Mastoid tip. D. Posterior wall of external auditory canal.

border of the parotid gland and seemed to extend into the region of the inner prolongation of the gland. It was impossible to outline the mass definitely, but it seemed to be about 4 cms in diameter. The tympanic membranes were of normal appearance and there was no hearing loss. Radiographic studies of the skull showed no abnormality except an unusually large left jugular foramen and large foramina in the base of the middle cranial fossa. These were thought to be congenital but, in the light of subsequent findings, may yet prove to be of serious significance.

On August 9, 1946, an operation was performed in an attempt to remove the tumor which was thought to be of parotid gland origin. An incision was made to expose the parotid gland widely.

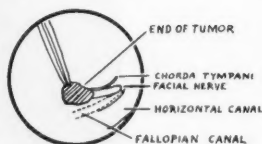
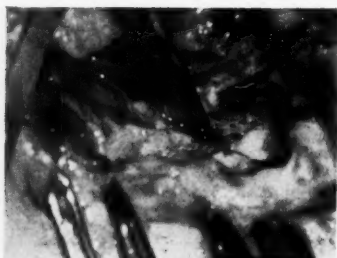


Fig. 9, Case 4.—Photograph taken during operation.

Fig. 9a.—Descriptive diagram of Figure 9.

There was considerable postoperative reaction in the tissues but the tumor could be palpated in the region of the inner prolongation of the parotid gland. An attempt to expose the facial nerve as it crosses the base of the styloid process,¹⁰ was unsuccessful. Nothing but tumor could be found adjacent to the styloid process. The zygomatic branch of the facial nerve was then identified and isolated in the upper anterior portion of the gland and traced cephalad. When this branch reached the tumor, it seemed to fan out in a thin wide ribbon in the tumor's capsule as did the other branches which were also isolated. It was in this apparent capsule that the thin wide nerve had been severed at the pes anserinus. It was rather obvious at this point that the tumor was a large solitary neurofibroma of the facial nerve. The tumor was then removed at the stylo-mastoid foramen which was found to be more than twice its normal size. The three branches of the pes anserinus previously isolated still had evidence of tumor in them. When the ends were resected to healthy nerve tissue, there were five separate distal branches. These were tagged and the wound was closed.

The second stage of the operation was performed on August 12, 1946. The mastoid tip was removed and the fallopian canal uncapped from below upward. It was hoped that the tumor would end in the vertical course of the canal and that a radical mastoidectomy with resulting hearing impairment could be avoided. In the upper portion of the vertical course of the fallopian canal the nerve was still greatly enlarged by tumor (Fig. 8). The technique of radical mastoidectomy was then completed and the facial nerve was exposed and mobilized to its point of entrance into the middle ear. The end

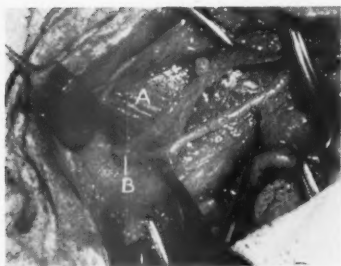


Fig. 10, Case 4.—Photograph taken during operation. *A.* Sterno mastoid muscle. *B.* Great auricular nerve before removal.



Fig. 11, Case 4.—Photograph taken during operation. *A.* Single trunk sutured to the proximal stump of the facial nerve in the middle ear. *B.* The five ends of the graft sutured to the five isolated branches of the facial nerve in the cheek.

of the tumor was visible grossly in the mid portion of its intratympanic segment (Fig. 9 and 9a). Excision was carried well into healthy appearing nerve.

The mastoid incision was then extended inferiorly and the great auricular nerve was isolated (Fig. 10). This nerve was large and branched behind the sterno-mastoid muscle. A single trunk with five branches measuring 6.5 cms in length was obtained for use as a free graft. The main trunk was sutured to the stump of the facial nerve in the middle ear and the five branches were sutured to the five distal branches of the facial nerve which had been tagged at the previous operation (Fig. 11).

The postoperative course was uneventful.

The histopathological diagnosis was cirroid neurofibroma. The crushed end of the specimen indicated complete excision.

On April 10, 1947, (eight months after operation) the patient noticed a little movement about the corner of his mouth. During the next month there was considerable improvement. When he returned on May 23, 1947, mass motion was present to a limited degree. The muscle tone was good and the patient could close his eye almost completely. He could move both the upper and lower lips but could not raise his brow. An audiogram showed an average of 45 decibels loss in the critical frequencies.

On September 12, 1947, further improvement was noticed as demonstrated in the photographs (Fig. 12).

This case is deserving of particular comment due to the apparent rarity of solitary neurofibromas of the facial nerve. In a cursory review of the literature several reports have been found dealing with solitary facial nerve neurofibromas of the Schwannoma or Antoni A type and classified as neurinomas. Lundgren,¹¹ in 1947, reviewed the literature on this subject and reported one instance in which the tumor was found in the external auditory canal lateral to the tympanic membrane. No facial paralysis was present. At subsequent operation the tumor was found to have originated from the vertical portion of the facial nerve. Altmann,¹² cited by Lundgren,¹¹ suggested that the first and only symptom for a long time, perhaps years, was facial paralysis with later hearing impairment as the middle ear was invaded. Kettel,¹³ cited by Lundgren, was able to collect information on 17 patients with facial nerve neurinomas.

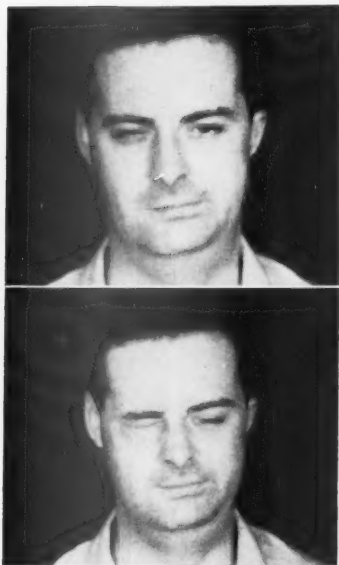


Fig. 12, Case 4.—Photographs taken 13 months after nerve repair.

Again, facial paralysis seemed to be a characteristic finding. He reported removal of one tumor from the vertical course of the nerve and used a nerve graft for repair. He commented on the rarity of neurinomas in motor nerves and suggested that facial neurinomas might originate from sensory fibers in the facial nerve. Williams,¹⁴ in 1939, reported a case of neurofibroma of the facial nerve involving the mastoid process and extending into the petrous. Facial paralysis had been present for four years. Roberts,¹⁵ in 1943, and Bogdasarian,¹⁶ in 1944, each reported cases of solitary neurinoma of the facial nerve in which there was preoperative facial paralysis.

Unlike the other reported neurofibromas of the facial nerve, the one presented here was of cirroid or plexiform type. Insofar as could be determined, the tumor was solitary but extended into the distal branches in a characteristic manner. In two other instances I have seen the facial nerve involved extensively by a plexiform neurofibroma without any impairment of function. In each instance,

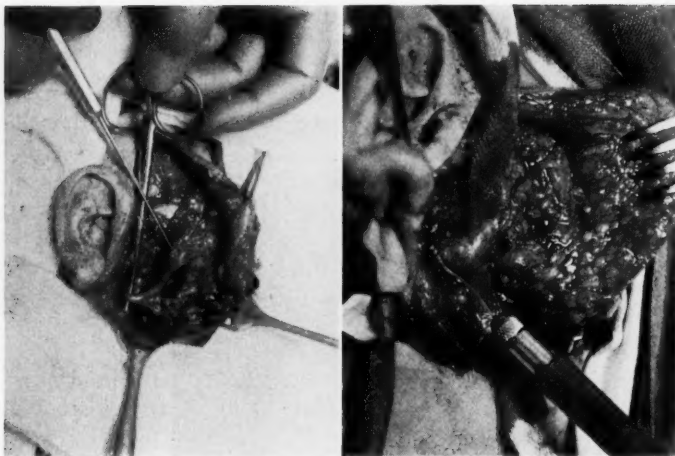


Fig. 13, Case T. G. 492222.—Photograph of facial nerve taken during operation.

Fig. 14, Case L. G. 682889.—Photograph of facial nerve taken during operation.

the patient was a small child on whom operation was performed for removal of a tumor below the angle of the mandible and apparently extending into the parotid gland. In each one, the tumor proved to be multiple neurofibromas of the sensory cervical nerves but also involved the facial nerve. They were classified as von Recklinghausen's disease. The appearance of the facial nerve in each case is shown in Figure 13 and Figure 14. The first patient was a white male, T. G., aged 2 years. The operation was performed on September 25, 1945. The second patient, L. G., was a white male, aged 19 months. Operation was performed on February 28, 1951.

Although there was no facial paralysis in the three cases of cirroid neurofibroma of the facial nerve cited here, it would seem quite likely that facial paralysis would be produced if the tumor were to originate within the confines of the fallopian canal as in the reported cases of neurinomas. The gradual stretching of the nerve in the soft tissues is not apt to produce paralysis. It does seem remarkable, however, that complete function of the facial muscles had been

retained in the patient whose neurofibroma extended through the entire vertical course of the fallopian canal.

SUMMARY

Four cases of facial nerve repair have been reported. These were not selected to demonstrate excellent return of function, but rather to show examples of particular problems which may be encountered by the surgeon, and the different methods of repair which may be utilized.

In each case, repair involved the facial nerve within the parotid gland although in three instances this was combined with intra-temporal repair. In three of the cases, repair was tantamount to reconstruction of the pes anserinus.

In addition, three cases of cirroid neurofibroma of the facial nerve without facial paralysis have been recorded.

UNIVERSITY HOSPITAL

DEPARTMENT OF OTOLARYNGOLOGY.

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DISCUSSION

DR. EDGAR P. CARDWELL: Thank you, sir.

Mr. President, Members, and Guests of this distinguished organization:

I have a short motion picture film to show the result following the removal of a tumor with a large section of the facial nerve and its repair. There is some question about the tumor being a benign tumor of the parotid. There was no paralysis before the first operation.

If you will start the film please, I will continue so I can have all the time allowed me to talk about this important case. This patient was 22 years old. He had a parotid tumor for about three years. He was operated upon April 20, 1936, and the large tumor removed with the facial nerve incorporated in the mass. On April 23, three sections of the anterior femoral cutaneous nerve were sectioned in order to cause degeneration of these nerves. On April 27, these three prepared grafts, 60-70 mm long, were laid in the reopened wound in place of the nerve removed with the tumor, together with four freshly cut nerve grafts of the same length.

While the film runs, I would like to give you the rest of the story on this man. On the 27 of April, grafts were laid in, attached by a single suture, to the freshly sectioned nerve trunk. At the end of about six months there was some electrical response. Shortly after that there was a partial recovery of lower face function. At the end of a year, (he was very hard to keep track of, as he did not want to come in) he had a very good function of the lower two-thirds of his face, but I think that you will notice in this film, that when it was taken, two years after the operative procedure, he had good frontalis function. That is the scar of the operation beside his ear.

The peripheral ends of two of the degenerated grafts were laid alongside of two marked terminal nerves, which were too small to hold sutures. The other three freshly cut nerves had their peripheral ends laid directly in the temporalis and orbicularis muscles, respectively, with no effort to locate terminal nerve trunks.

Allow me to thank Dr. Maxwell for the opportunity of showing this picture to you gentlemen. I hope that you will be encouraged to help these poor people, many of whom are going around the country with terrible looking faces. In fact, they have lost half "the mirror of their souls."

Reference: Cardwell, Edgar P.: Direct Implantation of Free Nerve Grafts Between Facial Musculature and Facial Trunk, *Arch. Otolaryngol.* 27:469-471 (Apr.) 1938.

PRESIDENT DAY: The discussion will be continued by Dr. Sullivan.

Dr. Sullivan!

DR. JOSEPH A. SULLIVAN: Mr. President, Members of the Otological Society, Honored Guests, Dr. Maxwell in his usual clear and precise manner has given us a series of cases today that I believe as Otologists we are fully aware that this condition exists but he drives it home to the general surgeon that the facial nerve does run through the parotid gland. It makes its exit at the stylomastoid foramen and some effort should be made to preserve it.

In the cases that he has presented to us he apologizes for the return of function. I think they are excellent.

In any type of facial paralysis we aim at two functions: one, the ability to close the eye, and secondly, the ability to elevate the corner of the mouth. If a patient obtains that result, then he gets along very well in life.

I believe the last case that Dr. Maxwell demonstrated is really an exceptional case. He mentioned the use of a single graft in preference to a cable graft. I might add a word, that I believe that is the reason he got such a good result.

The moving picture presented by Dr. Cardwell, if I am correct, in those cases was there an attempt to plant the nerve directly in the muscle tissue?

DR. CARDWELL: There was.

DR. SULLIVAN: That is a remarkable picture and it is a remarkable fact because it revolutionizes the neuro-anatomical junction of the nerve in the muscle.

A number of years ago we experimentally tried to implant nerve into muscle without restoration of function. We did not have any results and it does contradict the work of Cahal and the neuroanatomists in that particular regard.

I might add a further word in regard to Dr. Maxwell's presentation, that this type of lesion external to the stylomastoid foramen, in some of the doubtful cases. At the time of direct repair of nerve it is a good idea at the same time to use some form of sling-up operation or the use of a silver wire. I think that is an important point in these types of peripheral injury, extratemporal.

Again I would like to congratulate Dr. Maxwell on a most excellent preparation.

PRESIDENT DAY: Is there any further discussion of Dr. Maxwell's paper? Dr. Muerman!

DR. OTTO MEURMAN: Mr. Chairman and Gentlemen I want to congratulate Dr. Maxwell for his excellent results.

I just have one remark to make here about the time of the recurrence of the first movements. I had recently a case of quite a young child, I suppose she was about five years, in which there was a defect of about two centimeters within the mastoid, and of course I replaced that with a graft of the same length. The first movements came not quite five months after the operation.

I just wanted to call your attention to that, that presumably in those children the movements come back earlier than in adults. I had an experience with children before but this was remarkable how early they came back. I did not quite

hear how early the movements came back to Dr. Maxwell's case. Was it six months or before in that boy?

DR. MAXWELL: In the small child five months when he first noticed it.

DR. MEURMAN: It corroborates then.

Again as you remember the proposal made by Bellans-Newell, to take the external cutaneous, not to replace the facial node, I think that that is not quite successful as a proposal because the external femoral nerve is very thin and in some cases they actually did not tie. Then I did as you did.

Several years ago I began to take the post-auricular node, which is a very, very good one to replace and I think that there may be some importance to have as thick a graft as possible because there ought to be sheath enough for the neural fibers to grow in.

Thank you very much!

DR. GEORGE L. WHELAN: Gentlemen, I am sure we all appreciate Dr. Maxwell's excellent presentation. If we have not been impressed about the seriousness of face tumors, of the facial nerve and about the face we should now be.

I should like to have Dr. Maxwell tell us briefly in closing just how he is able to identify the nerve and pick it up. If we are under the impression that we can pick up by sight the small branches of the nerve in muscular tissue from the depths of the wound I believe we are mistaken. I think Dr. Maxwell has an improved method. Perhaps the rest of you are familiar with it but I would like to have him say a word about it.

DR. JAMES H. MAXWELL: I want to thank those gentlemen who discussed my paper. Dr. Cardwell's movie was most interesting and I wish to thank him for showing it at this time. I was particularly interested in this case because, as I remember the published report—Dr. Cardwell, please correct me if I am wrong—three branches of the anterior femoral cutaneous nerve were sutured to the proximal stump of the facial, and then because all the distal branches tagged at an earlier operation were not discernible, these grafts were laid next to those distal branches that were found without suturing. Then in addition, other branches of the anterior femoral cutaneous nerve were sutured to the proximal stump of the facial and their distal ends implanted in the muscles. I wonder if your conclusion that the functional result was due to the implantation of those nerves in the muscle was correct. I would be rather inclined to think that you might have had regeneration from those branches that you laid next to the distal branches. Otherwise, regeneration of the motor end plates would be required. It has been my understanding that motor end plates are not capable of regeneration.

DR. EDGAR P. CARDWELL: There were only two of the peripheral branches that I could locate at the operation for nerve grafting. The other nerves were fanned out, roughly in the direction that I had remembered the branches coming off from the tumor, in little tunnels made with a small Hemostat into the muscle. There was no real indication as to where to put the other branches, except that I directed the two grafts toward the orbicularis muscle and two grafts up toward the frontalis muscle. Apparently, all the grafts have resulted in function.

DR. MAXWELL: It was a most interesting case and you are to be congratulated on the splendid results.

In regard to Dr. Sullivan's remarks, I really was not apologizing for the results in these cases. I was just wishing that it had been possible for me to obtain better results. I have never obtained a result in one of these cases with which I was thoroughly satisfied.

The suggestion of using a sling at the time of the original operation is an excellent one. It gives support to the muscles during the time of regeneration which is so important.

Dr. Muerman asked about the time required for regeneration. In the case in question, the repair was peripheral and the axones did not have as far to go as they would have if repair had been within the temporal bone. It would seem to me that that might have accounted for a little more rapid return of function than was noted in some of the other cases. I have not noticed any significant difference between children and adults insofar as time required for regeneration is concerned. I have noticed, however, that in those instances in which recovery was very slow, such as eight or nine months, my results were poor.

In regard to the selection of the donor nerve, I prefer the great auricular because it is so conveniently accessible. Also, it is rather firm and does have the gelatinous nature of the anterior femoral cutaneous nerve which facilitates suture when in an unprepared state. Dr. Sullivan has spoken many times on the desirability of using prepared grafts and undoubtedly there are some advantages in this procedure. In these particular cases I did not use prepared grafts for the very good reason that after the distal branches had been isolated with great difficulty it seemed hazardous to bury them again for two weeks before inserting the grafts.

When the pes anserinus has been disrupted, a satisfactory first procedure is the isolation of the zygomatic branch which parallels the parotid duct about a finger's breadth below the zygomatic arch. This may then be traced cephalad to the region of the pes.

Dr. Bunnell in one of his earlier publications on this subject made an excellent suggestion about following the small branches distally. He advised locating the proximal segment at the point of interruption and then inserting a fine needle in the general direction of the distal branch. This distal branch may then be located by dissecting down on the needle.

POSTURAL VERTIGO AND POSITIONAL NYSTAGMUS

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The complaint of vertigo or dizziness which occurs when certain postures or positions are assumed and is absent in others is known as a "postural vertigo." The dizziness is of the vestibular type, described usually as a sense of movement or whirling of the environment or the subject. It is accompanied by a "positional" nystagmus as well as ataxia and vegetative reflexes depending upon the severity and duration of the symptom.

Inasmuch as dizziness is a subjective sensation and therefore not always susceptible to accurate evaluation it is preferable to discuss the syndrome in terms of the objective sign, the positional nystagmus. The occurrence of this positional nystagmus in association with the complaint of dizziness establishes the presence of a disturbance of the vestibular apparatus.

Postural vertigo has been the subject of considerable investigation, both laboratory and clinical, in various European centers in recent years but has not received similar attention in America. The frequency with which dizziness of great severity occurs only in certain postures, and the necessity of specific questioning to elicit this postural character have not been fully realized. The routine postural tests which are necessary in order to reproduce the syndrome and provide objective evidence of the disturbance have not been regularly included as an essential part of the examination of the vestibular mechanism.

The investigations of Ewald,¹ Mach,² Breuer³ and Crum-Brown⁴ before the turn of the century and later the animal experiments of Magnus and de Kleyn⁵ concentrated attention on the otoliths as the receptor organs concerned with linear acceleration. The occurrence

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of vertigo and nystagmus when the head was placed in certain positions was at first thought to indicate a disturbance of the otoliths hence was known as otolith nystagmus.^{6,7} As clinical evidence has accumulated it has become evident that a postural vertigo may indicate a disturbance of either the central or the peripheral vestibular apparatus.

It is possible that a postural vertigo that comes from an inner ear disease may originate in the otoliths but it has so far not been possible to obtain proof. The otoliths have been shown to cause counter-rolling movements of the eyes in lower animals^{8, 9} but the question as to whether they may cause a nystagmus remains undecided.

A postural vertigo and positional nystagmus of central origin has been produced experimentally after bilateral labyrinthectomy¹⁰ hence the phenomenon does not depend upon the presence of a functioning receptor organ.

The clinical importance of a postural type of vertigo does not depend upon which type of vestibular sense organ is affected. In the present state of our knowledge the primary significance lies in the fact that the positional nystagmus which accompanies the complaint affords proof of an organic disease of the vestibular mechanism. The information provided by the characteristics of the nystagmus as to localization is frequently of definite value but the diagnostic implications of the various types have not yet been fully explored. It is of practical importance that a postural vertigo may escape detection if specific questions are not asked to elicit such information and a positive history followed up by examination for positional nystagmus. The postural tests involve movements which are common in everyday life and the effect on the vestibular mechanism comes within the range of normal physiologic stimulation as compared to caloric tests which utilize non-physiologic stimulation and turning tests which, when carried out in the customary way, constitute overstimulation.¹¹

DEFINITIONS

Because of the many variations in position, and changes in position which may bring on the syndrome of postural vertigo and positional nystagmus some difference of opinion exists as to what should be considered as "postural" or "positional."

From the practical point of view it is probably of greatest value to differentiate between a "spontaneous" and a "positional" nystag-

mus. A spontaneous nystagmus is one which is present in the normal resting position, usually the erect position, and which continues to be present in similar intensity regardless of alteration in position of the head.

The definition of a "positional nystagmus" is less simple because of the many variations which occur.

For practical reasons it is considerably preferable to include as "positional" all cases in which the nystagmus appears when certain positions are assumed and is absent in other positions. Within this general group may be distinguished several sub-groups which will be referred to under "classification."

A "positional" nystagmus is differentiated from the nystagmus which in some cases may be activated by the head shaking test. The head shaking test and the postural tests have no definite relationship. The former provides an abnormally strong stimulus which tends to activate a "latent" spontaneous nystagmus, but usually does not bring out a "positional" nystagmus. The postural tests consist of physiologic movements to bring the head and body into a series of postures or positions and do not tend to activate a latent nystagmus. Both tests may however give a positive response in the same disease of the vestibular system.

A positive fistula reaction should also be differentiated, as well as the nystagmus which in rare cases may be produced by applying pressure over the vessels in the carotid sheath.

METHOD OF EXAMINATION FOR POSITIONAL NYSTAGMUS

Although the postural nature of vertigo or dizziness is seldom indicated in the patient's description of his symptoms a few specific questions will bring out this characteristic in detail since patients become acutely aware of those positions which they must avoid. In carrying out the ear examination postural tests are best preceded by the routine otoscopic examination, hearing tests, examination for spontaneous nystagmus, exclusion of a labyrinthine fistula, the effect of pressure over the carotid sinus region and the head shaking test.

In order to avoid neck reflexes the postural tests should theoretically be done with the head and body in the same relationship.

In the human, however, the neck reflexes have a negligible effect on eye movements and do not complicate the tests.

Special tables have been constructed by Grahe¹² and Fromm and Nylen¹³ to permit the patient to be passively shifted into all postures and to allow photography of eye movements during the tests. For clinical purposes, however, an adequate series of tests can be carried out with the patient on an ordinary examining table.

Observations of the eyes for positional nystagmus may be done with or without the use of +20 diopter lenses to eliminate fixation.

The eyes are routinely examined in the position of forward gaze. Lateral gaze is not necessary, and even introduces a complication in those cases presenting a spontaneous nystagmus in the direction of gaze. A routine sequence in testing the various positions is advisable, and should be adhered to if possible.

While the patient's history indicates which positions are likely to yield positive results the completion of the whole series of positions is advisable since further information may thus be obtained.

The order in which the tests have been found to be convenient is as follows: 1. Sitting on the table in erect posture. 2. From erect to supine. 3. From supine to right lateral. 4. From right lateral to supine. 5. From supine to left lateral. 6. From left lateral to supine. 7. Head extended over the end of the table. 8. Supine position again. 9. From supine to erect. 10. Feet over the side of the table and stooping forward to the head-hanging position, and 11. From stooping over to the erect position.

The above positions and the movements used are all in common everyday use with the exception of "head extended over the end of the table." This position introduces possible factors of tension on neck muscles and circulatory interference.

An optokinetic nystagmus is always visible during the change in position and is even increased if fixation is eliminated by the use of the lenses.

A factor which is of special significance is the rate at which the positions are changed. In certain clinical conditions the positional nystagmus is independent of the rate of change in position. The nystagmus may be temporary or may be maintained while in that position. In other clinical conditions the syndrome may be dependent

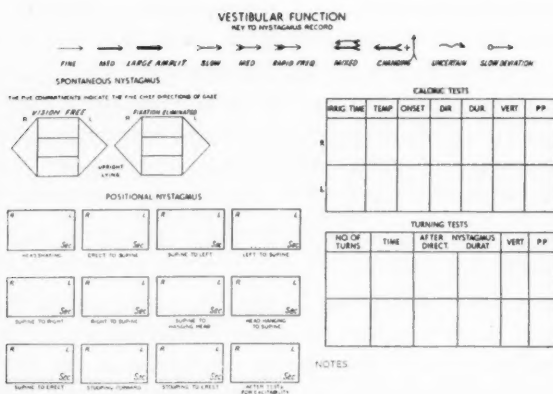


Fig. 1.—A simplified chart (modified from Frenzel) for recording spontaneous nystagmus, positional nystagmus and responses to caloric and turning tests. The two six-sided diagrams are divided into five compartments each to represent directions of gaze. Arrows are used to indicate direction amplitude and frequency of the nystagmus. The three rows of rectangles are used for recording positional nystagmus and are arranged in the order from left to right in which a series of routine postural tests may be carried out with the least difficulty on an examining table.

on the rate at which the new position has been reached although the syndrome may appear either before or after the new position has been completely attained. This latter type is in the author's experience one of the most frequent seen in office practice and includes many cases where the underlying etiology is related to vascular or vasomotor insufficiency as well as many cases of vertigo following head trauma. In such cases it may be necessary to change the position at a certain rate in order to bring on the vertigo and nystagmus. The rate of movement however still conforms to that which is common in everyday activities. Objection has been raised to the changing of position quickly since the reaction may then be due to the "movement factor" rather than "position." It must however be pointed out that although a slow change of position may fail to elicit the symptom in certain cases, these subjects may experience severe vertigo if the movements are carried out at the rate which is customary and necessary in their daily routine activities. The inclusion of such cases under positional nystagmus is indicated for practical reasons.

The first objective of the postural tests is to reproduce the syndrome. Therefore if a slow change of position fails to do so, in the presence of a positive history the test must also be carried out more rapidly. The position may be changed slowly on the first test and if negative it may be repeated at a more rapid rate.

The recording of the findings on examination for spontaneous nystagmus as well as head shaking and postural tests can be conveniently recorded in the diagram shown in Figure 1 which is modified from that designed by Frenzel.¹⁴ The rectangles are situated in the order in which the postures are conveniently tested. It is indicated in each rectangle whether the particular posture was tested and the results.

A continuous recording of the eye movements has been made using a special position table with motion picture camera attached¹³ and can also be achieved by recording the corneo-retinal potentials during the tests.

The tests for vestibular excitability have been customarily made following the examinations for spontaneous and positional nystagmus. Of these, the cold caloric test, according to the principle of minimal stimulation while using +20 diopter lenses to observe the nystagmus, has been the basic test. When necessary the stimulus is then increased by lowering the temperature of the water.

CLASSIFICATION OF POSITIONAL NYSTAGMUS

The two basic questions that have excited interest in postural vertigo and its accompanying positional nystagmus¹⁵ have been the pathophysiologic basis for the syndrome, and the significance of positional nystagmus in otoneurologic diagnosis. Experimental investigations by de Kleyn and Versteegh,¹⁶ Nylen,¹⁷ Chilow,¹⁸ Hasegawa,¹⁹ Fromm and Nylen,¹⁰ Spiegel and Scala²⁰ have demonstrated that a positional nystagmus may be produced by several types of disturbance affecting either the peripheral or the central part of the vestibular apparatus.

Clinical observations by Nylen²¹ in cases of brain tumor, by Seiferth²² in other disease states as well as by Ruttin,²³ Frenzel,¹⁴ Lindsay²⁴ and others have demonstrated the frequency of the syndrome in certain diseases of both central and peripheral localization.

Several classifications of positional nystagmus have been made^{23, 25, 22, 15} which represent an effort mainly to assess localizing significance of the different types.

Of these classifications there has been some agreement as to two main types which appears to have some significance in differentiating a central from a peripheral lesion. If this grouping is adhered to it becomes at once apparent that there are certain well differentiated subgroups. It is probable that the characteristics of these sub-groups may provide significant diagnostic information.

Type I. Direction-changing positional nystagmus. (Nylen's type 1, Seiferth's type 1, Ruttin's type 1, Lindsay's type 1, Frenzel's "true positional nystagmus").

The direction is changed in certain positions of the head, usually being reversed in the opposite positions. It may, however, be horizontal in one position, for example a lateral position, and may be vertical, oblique or rotary in some other position.

Using the heading of a "direction changing" nystagmus it is possible to differentiate a regular or constant type and an irregular type. The irregular type presents several varieties. Thus sub-group (a) might be called "regular" and sub-groups (b) (c) and (d) variation of "irregular positional nystagmus." (Irregular positional nystagmus is classed as Type 3, by Nylen.²⁵ However, in view of the fact that all of these sub-groups exhibit the "direction-changing" characteristic either constantly or intermittently they are all listed here as sub-groups of Type I positional nystagmus.)

(a) Direction-changing positional nystagmus maintained. A common form is that in which the direction reverses in opposite positions of the head.

(b) Direction-changing but the positional nystagmus has a limited duration, usually less than one minute.

Occasionally in this type after the nystagmus has reached the end point it may then reverse its direction for a shorter period at much lower intensity.

Another variation of this type is that in which the appearance of the nystagmus depends upon the rate of alteration in position. For example, if the change from the supine to a lateral position, or

from supine to an upright position is carried out slowly the vertigo and nystagmus may not occur but if repeated a little faster, the syndrome may appear in its usual intensity and for the usual duration.

Since many clinical cases such as those due to head trauma or to vasomotor insufficiency may correspond to this type, the practical importance of repeating the test is evident.

A positional nystagmus of this type may sometimes show minor variations in its characteristics on repeated tests.

(c) Positional nystagmus may appear before the customary ninety degrees of change in position has been completed or when the change has progressed to a certain point in one direction. It may then be temporary in duration or maintained.

(d) Another variation or type of positional nystagmus which is usually of the "direction changing type" is that in which the positional nystagmus may only appear when the previously held position has been maintained for a considerable period of time. For example, a positional nystagmus on turning to a side position from the supine may fail to appear unless the supine position has been held for some minutes before turning to the side position.^{26,27} Such an irregularity is not necessarily confined to Type I.

Type II. Direction-fixed nystagmus. In this type the nystagmus may only appear in certain positions, but is constant in its direction. (Nylen's type II, Frenzel's spontaneous nystagmus released by position stimulus).

Two subdivisions may be distinguished in this group:

(a) No spontaneous nystagmus is present but a positional nystagmus occurs in one or more positions and is constant as to direction. Before a positional nystagmus can be classified in this group it is necessary that the routine postural tests be completed, and all positions investigated.

(b) A spontaneous nystagmus is present in all head positions, but an increase in intensity appears in certain positions, the direction remaining the same. In this type the posture influences the intensity of the spontaneous nystagmus. The inclusion of such cases under "positional" nystagmus is of questionable value since the vestibular disturbance is already indicated by the spontaneous nystagmus.

CLINICAL IMPORTANCE OF POSITIONAL NYSTAGMUS

The clinical evaluation of positional nystagmus is based upon the observations which have been made in cases of vestibular disturbance of known localization and etiology.

It is therefore convenient to consider the phenomenon in three main groups depending upon whether the disturbance can be localized to the central or peripheral vestibular system or is of doubtful localization.

Positional nystagmus in central vestibular affections. On the basis of clinical observations in diseases of the vestibular system of known localization a direction-changing positional nystagmus (Type I) has been found to be strong evidence of a central vestibular affection.^{14, 21-24} This is particularly true of cases in which the direction reverses in opposite positions. This cannot be considered a rule, however, since a limited number of examples of a "direction changing" positional nystagmus have been observed in peripheral vestibular disease.

A positional nystagmus of fixed direction has been frequently observed in both central and peripheral vestibular affections.

Those diseases of the central nervous system in which a vestibular disturbance is commonly present and in which the vertigo and nystagmus may be present only in certain positions include encephalitis, multiple sclerosis, brain tumor, abscess, hemorrhage, emboli or thrombosis.

In such cases the diagnosis usually is dependent upon the accompanying signs and symptoms related to the central nervous system, but cases of encephalitis and brain tumor have been observed in which at a certain stage a positional nystagmus has been the only demonstrable neurological sign.

Fromm²⁸ found that when postural tests were included a nystagmus was demonstrated in 90 per cent of multiple sclerosis and of these a positional nystagmus was found in almost two-thirds. In these the type of positional nystagmus corresponded to Type I in over 33 per cent and Type II in the remainder.

Nylen²¹ in a series of 673 verified cases of brain tumor demonstrated a positional nystagmus in 279 of the 360 cases with nystag-

mus. In 50 per cent the positional nystagmus was direction-fixed, in 30 per cent direction-changing and in 20 per cent the nystagmus was spontaneous.

In subtentorial tumors positional nystagmus of all types was found in 69 per cent of 240 cases, while in supratentorial tumors the incidence was 26 per cent of 433 cases. Nylen also observed that in tumors of the posterior fossa the positional nystagmus most frequently occurred in the side positions or many positions while in supratentorial lesions it was most frequent in the "hanging head" position.

A positional nystagmus is frequently found in toxic conditions, hypertension, hypotension, arteriosclerosis, vasomotor instability, head trauma and other states. In these conditions the vestibular disturbance is usually temporary, without detectable changes of permanent nature, and without signs that would with certainty localize the disturbance to the peripheral or central vestibular system. These conditions are therefore included under group (c) in which the localization is considered as uncertain.

Positional nystagmus in peripheral vestibular affections. (1) Inflammatory tubal and middle ear disease of the non-suppurative "secretory" type is occasionally accompanied by vertigo, and this may be affected by posture. A direction-fixed positional nystagmus appears to be most common.²⁹ Relief may be afforded by removal of the fluid from the middle ear.

(2) The occurrence of positional nystagmus in suppurative otitis media, labyrinthine fistula and labyrinthitis has been frequently reported.³⁰

(3) Ménière's disease or labyrinthine hydrops of the idiopathic type: Ménière's "syndrome" has been reported as being frequently accompanied by a positional nystagmus. In the writer's experience the incidence of a positional nystagmus in typical Ménière's disease, in which the predominating evidence indicates the presence of a hydrops of the labyrinth, bears a striking contrast to that in "atypical" or "pseudo" Ménière's syndrome. The latter is differentiated from hydrops of the labyrinth by the absence of the classical auditory phenomena. The positional nystagmus provides a further significant point of differentiation.

In the typical Ménière's disease or labyrinthine hydrops, during an attack when a spontaneous nystagmus is present in all positions, the intensity may be influenced by posture. The inclusion of such cases under "positional nystagmus" materially increases the incidence in that disease. In the stage of Ménière's disease where there is only a complaint of unsteadiness, but no spontaneous nystagmus the head shaking test may activate a latent nystagmus, but the occurrence of positional nystagmus is not common. The more characteristic pattern of the vertigo in Ménière's disease is a more or less rapid onset followed after a varying period of time by a more or less rapid subsidence of all vertigo.

In contrast to Ménière's disease (labyrinthine hydrops) a positional nystagmus has been observed so frequently in "pseudo" or "atypical" Ménière's syndrome that it may be considered as a characteristic. A direction changing (Type I) positional nystagmus has been most common. Another feature of the positional nystagmus in this group is its long duration, varying from a period of days or weeks to months.

(4) Postural vertigo associated with deafness of sudden onset: A type of inner ear disease which has usually been classed as Ménière's syndrome" and sometimes confused with "Ménière's disease" is that in which there is a sudden onset of inner ear deafness primarily for high tones and vertigo in an ear which has previously been healthy.³¹⁻³⁴

The occurrence of this syndrome in all periods from the third decade of life onward, without clear cut evidence of associated disease, has left the etiology undecided.

The damage to inner ear function varies greatly and in a high percentage the vestibular involvement appears to bear a relation to the degree of hearing loss. Thus a mild hearing loss for high tones only may be accompanied by little if any vestibular involvement. A profound hearing loss may be associated with severe vertigo of limited duration and a loss of vestibular responses. However, a moderate loss for high tones is often accompanied by more or less severe vertigo for a few days followed by postural vertigo for as long as three years or more, with normal or slightly depressed vestibular responses.

The positional nystagmus observed in this group has been of the "direction changing" type, lasting usually less than one minute,

but reversal of the nystagmus in opposite positions of the head has not been observed.³¹

Positional nystagmus in diseases with uncertain localization. The available evidence points to a central localization in most of the conditions discussed under this group, but the proof is lacking. In this group of cases the complaint of dizziness is not accompanied by tinnitus or any change in the hearing and the examination reveals no conclusive signs of disease of the central nervous system. It probably constitutes the majority of cases seen in clinical practice.

In this group it is frequently possible to associate the symptom with a systemic state or disease including intoxications from drugs or infections, arteriosclerosis, hypertension, hypotension, vasomotor instability, menopause, allergic states or head trauma. In many instances there may be no evidence as to etiology.

Certain drugs, for example streptomycin, may affect the vestibular nuclei primarily, others such as quinine and salicylates appear to affect the peripheral ear since auditory and vestibular phenomena occur together without CNS signs, but drugs such as morphine, alcohol³⁵ barbiturates and others may cause a postural vertigo and positional nystagmus without auditory symptoms or measurable impairment of vestibular function.

Infections of the sinuses, the respiratory tract in general or the intestinal tract may cause a vertigo without auditory symptoms. The vertigo is typically postural and usually of Type I.

In arteriosclerosis and hypertension dizzy spells may occur without other symptoms and, although a spontaneous nystagmus may be present at the onset, the syndrome frequently becomes postural within a day or two and persists as such for a few weeks.

In hypotension, vasomotor instability and the menopause the syndrome is characteristically postural throughout and the positional nystagmus usually of the "direction-changing" type (Type I). In these states the postural tests may be influenced both by the rate of change of position, the particular posture which precedes that in which nystagmus occurs, and the period of time spent in the previous posture.

Head trauma is recognized as a common cause of dizziness in subsequent months. The dizziness is characteristically postural and varies

in severity.^{22, 30} The postural tests may have to be repeated more rapidly to elicit the positional nystagmus in some instances. A "direction-changing" nystagmus is most frequent and suggests a disturbance in the vicinity of the vestibular nuclei.

In those instances where the postural vertigo has apparently been associated with one of the above diseased states the usual finding on caloric stimulation has been an approximately normal response, which corresponds with the temporary nature of the disturbance although duration of the syndrome for several months is common.

The finding of a marked asymmetry in caloric responses must be considered as suggestive of a more extensive lesion or of a progressive disease.

PATHOGENESIS OF POSITIONAL NYSTAGMUS

In peripheral affections: The positional nystagmus observed in acute inflammatory states customarily occurs in the early stages of inner ear involvement according to experimental and clinical observations and suggests a physico-chemical or pharmacological change in the labyrinthine fluids. If the inflammatory reaction increases in severity a spontaneous nystagmus appears. The positional nystagmus thus may represent an initial stage in the development of a diffuse labyrinthitis.

In cases of "secretory otitis" the inner ear reaction may remain in the initial stage probably because the inflammatory process in the middle ear is sterile and the effect on the inner ear remains limited in degree.

The positional nystagmus which occurs and persists for months or years following a single labyrinthine disturbance (sudden deafness of inner ear origin) requires a somewhat different explanation. A disturbance in a vestibular receptor of a more permanent nature seems a plausible explanation.

The experiments of Nylen, Chilow, Hasegawa, de Kleyn and Versteegh, have demonstrated that a positional nystagmus may be produced experimentally by inner ear disturbances of several types but it has not been possible to determine which type of vestibular end organ has produced the reaction.

In central affections: Animal experiments have shown that a positional nystagmus might be produced by lesions of various parts of the central vestibular system.^{20, 36, 10}

It is known that it may be produced without participation of the peripheral sense organs, and may arise from the vestibular nuclei, and from the cerebellar connections of the vestibular system. Clinical observations indicate that it may be produced by a number of diseased states, including an impairment of vascular supply or a local vasomotor disturbance or instability, intoxications involving certain nerve elements and the pressure affect of tumors.

The vestibular centers, primarily the nuclei may be considered as normally in a state of equilibrium. It has been suggested by Nylen²⁵ that the presence of a diseased state affecting one side or both sides unequally might result in an abnormal response to a normal peripheral stimulus. Such an abnormal response may occur also without participation of the peripheral sense organs.

It appears therefore that the central vestibular system may be influenced not only by the postural pressure effect of a space occupying lesion such as tumor, but that under normal conditions the central vestibular system is not completely immune to an effect from change in position. In the presence of a diseased state the ability to compensate for positional changes may be impaired, probably through deficient circulatory or vasomotor compensation.

Such a deficiency in circulatory compensation may directly affect the metabolism of the nerve cells and thereby create a disturbance of tone in the system.

THERAPY

Positional nystagmus is a symptom of different types of conditions affecting the vestibular system. Therapy is directed primarily at the etiologic process.

In the event that the presence of a definite systemic or local disease cannot be established the treatment may be limited to an attempt to relieve the symptoms.

The patient who first experiences a dizzy spell has two main concerns, the relief of the acute symptoms and the question of prognosis.

Relief of symptoms of postural vertigo is limited mainly to avoidance of positions which bring on the disturbance. Barbiturates and other drugs which appear to alleviate motion sickness may be of value.

When dizziness is postural in character a specific history regarding the effect of various positions and a routine set of postural tests not only provide factual objective evidence but give the patient assurance that his problem is appreciated. While the treatment is usually directed to the etiologic disease, many cases are encountered in which an etiologic diagnosis cannot be made with certainty.

In such circumstances the exclusion of a progressive disease either of the C.N.S. or of the inner ear warrants a good prognosis and reassurance for the patient. Such reassurance when based upon an adequate examination is an important part of the treatment.

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DISCUSSION

DR. ROBERT J. HUNTER: This is a very interesting paper and I think Dr. Lindsay is to be congratulated in that he has brought together a great many facts about this subject that have been scattered in different parts of the literature. One seldom hears a paper on this particular topic, so I think it is an excellent thing to have one today.

There are one or two points that I would like to ask about. I have found some cases in which once you induce this positional nystagmus, it is not possible to induce it again for a period of time. I had a patient, for instance, that complained that when he got up to speak, he was dizzy but if he held on to the lectern and kept his stance, he would recover in time. It sounds of course as though that was some circulatory affair but on examining him I found that when you changed his position from lying down to sitting up, he would develop spontaneous nystagmus, which had not been present when I first saw him. Once having induced that, if you tried to repeat that test, he didn't have it. After sending him out for a walk and asking him to come back in half an hour, you could elicit it a second time.

This patient had signs of toxemia, and repeated attacks of tonsillitis. Tonsillectomy seemed to cure him. Whether that was the source we do not know. I would like to ask Dr. Lindsay if he has any suggestion as to what the cause of this syndrome might be.

There is another form of positional nystagmus that the doctor mentioned that we see after severe illnesses. I recall a case, for instance, of an elderly lady with a bad attack of diphtheria, who had been given a great deal of antitoxin. You could demonstrate nystagmus when she assumed a certain position in the bed. I think those cases are much more common.

There is a third sort of case that Dr. Lindsay may have had in mind which he mentioned in a general way when he said that some of these cases are the result of head injuries. I have seen one case that I demonstrated to Barany when he was in this country which I reported under the title of "Disease of the Otolith Apparatus." [J. A. M. A. 78:1380-82 (May 6) 1922] One might say it was the mirror picture of cases he has been telling us of today. A child had a head injury. He had spontaneous nystagmus unless he put his head in a given position. The child would tilt his head to one side and elevate his chin as if bringing the horizontal canal in position and finally assume a spot where his eyes were stable. It seems to me now that it may have been a case in which the gaze apparatus was affected rather than the distinctly vestibular form of nystagmus. I would like to ask the doctor if he can give us any thought as to what could be the *modus operandi*.

I repeat myself by saying again that I enjoyed the paper very much.

PRESIDENT DAY: Dr. Shuster.

DR. BENJAMIN H. SHUSTER: Mr. Chairman and Gentlemen, I do not think Dr. Lindsay needed to apologize for bringing the subject to our attention again. There has been a dearth of work on that and Dr. Lindsay has probably done more than anyone in this country, clinically, along that line of work.

However, there are a number of things that did create some thought in one's mind. Whether the classifications that are laid out really have the important significance clinically is open to question.

The elicitation of positional nystagmus in itself is of importance so that we may be aware of its existence and not misinterpret it as a sign of an intracranial lesion when the vestibular tests are performed.

As far as the various classifications are concerned in listening to Dr. Lindsay, it almost seems like a cross-word puzzle. There are so many types of nystagmus, so many causes that one hardly knows exactly to what to attribute its existence. I do not think there is an apology necessary for this cross-word puzzle either because, like all cross-word puzzles, many of them sooner or later will assume definite patterns and will point to a definite lesion which in time will prove its importance.

I am not altogether sold on the various classifications that have been written in the literature, as Type 1, Type 2, one indicating an intracranial lesion and the other a peripheral lesion. First of all, it is nice to discuss and state that such-and-such a nystagmus is suggestive of intracranial lesion. Should you decide on the basis of that, to operate on a patient then that is another question. If one is going to do something about it, it is another matter.

Before we make a diagnosis of an intracranial lesion by merely changing the position of one's head, you want to say, "Suppose that were my head and the doctor wants to operate on me. Is he justified to operate on that particular point alone?"

Personally, I do not believe that very many cases showing positional nystagmus have any intracranial lesions. I believe most of them are due to peripheral irritability of the labyrinths and for that reason you get the reactions that you do. Almost any type of nystagmus that you can claim due to an intracranial lesions by changing the patient's position has been demonstrated to also be due to some peripheral toxic irritation.

For instance, it was thought and probably still is, that when one tilts the head forward and develops a vertical nystagmus it is almost certain that the patient has a posterior fossa lesion irritating the superior colliculi stimulating the centres for vertical eye movement and therefore there is a tumor of the posterior fossa.

Some years ago George W. MacKenzie had two cases of that sort which he reported in the literature in which nystagmus appeared after bending the head forward. After he had removed some infected teeth the positional nystagmus disappeared, so there was no tumor.

As to Nylen's description of various tumors of the head causing nystagmus, I had occasion in a busy neurological clinic to do tests on many patients with intracranial tumors. It is only seldom that one elicit positional nystagmus in cases with positively proven brain tumor cases. My opinion is that vast majority of cases showing positional nystagmus are peripheral. Of course there must be a number of cases which are caused by some intracranial lesion but are not necessarily a characteristic type. The nystagmus produced often follows a pattern similar to that which could be elicited by vestibular testing.

For instance, one can put the head backwards and develop a vertical nystagmus if both vertical canals are more irritable than the two horizontal ones. In this instance, one gets, actually, a direction of the current away from the cupola. You develop a nystagmus vertically down. One can figure out many kinds of nystagmus on the basis of current in the endolymph and conclude that most of those conditions are of a peripheral origin. The fact that one can elicit nystagmus objectively by changing head position implies that the patient may have vertigo,

which one may question, if the patient's veracity in stating that he has vertigo is questioned. This could be used as some objective proof of the patient's vertigo.

PRESIDENT DAY: Is there any further discussion of Dr. Lindsay's paper? If not, I shall ask Dr. Lindsay to close.

DR. JOHN R. LINDSAY: I want to thank Dr. Hunter and Dr. Shuster for discussing this question.

Regarding the first case mentioned by Dr. Hunter, a similar case was reported by Barany in the early twenties. In his case, after reproducing the vertigo and nystagmus once, it was then necessary to wait a half hour before it could be reproduced again. It is one of the variations that is sometimes seen.

Nystagmus after severe illness is not an uncommon finding. We don't know the explanation exactly. It could be central or it could be peripheral in origin. I think it is particularly important for us to recognize that the patient complaining of dizziness after head trauma may only have dizziness when he gets in a certain position. If he has an occupation which involves stooping or getting in unusual postures while, for example, working on a scaffold it might be dangerous and should be considered as some degree of temporary disability.

I thank Dr. Shuster for bringing up additional points since in the time allotted it is impossible to cover all phases of this question.

The point should be stressed that in these cases where there is a definite postural vertigo and positional nystagmus but no auditory symptoms and no other definite signs of neurological lesion, it is usually not possible to say whether it is peripheral or central in localization. Fortunately it is usually of little consequence.

The same applies to the inner ear, and the type of sense organ involved. It is of little practical consequence whether the otoliths or the canals are the source.

I think the point should be emphasized that one cannot make a diagnosis of a brain tumor or multiple sclerosis on the basis of the positional nystagmus alone. There is however the occasional case in which that may be the early symptom.

I would like to make one other point. It should not be assumed that a patient who complains of dizziness yet does not have a spontaneous nystagmus while sitting in the chair has a dizziness of psychic origin unless the question of postural vertigo has been investigated.

LONG-TERM RESULTS OF FENESTRATION SURGERY

HOWARD P. HOUSE, M.D.

LOS ANGELES, CALIF.

An analysis of our two-year results, together with a study of the causes of failure following fenestration surgery, was presented at a symposium before the Otosclerosis Study Group last fall. Following that meeting, your President asked me to analyze in a similar way the cases of patients who had had the fenestration operation five years or more ago.

A questionnaire is routinely sent to fenestrated patients six months, one year, and each year thereafter, following surgery. The information obtained in this manner is, I believe, of value in correlating our end results.

We have performed a total of 200 fenestrations which are now five years old or older. Unfortunately, we have been able to obtain complete records, including questionnaires and audiograms, in but 88 cases, representing 44 per cent of the total. In spite of every effort, 56 per cent of our patients are unavailable for accurate study after five years, a truly disappointing fact. This report, therefore, will deal with the 88 cases upon which we have complete records.

In studying our 200 patients I found that they fell into three general groups. (1) Eighty-eight patients who had returned a five-year questionnaire and on whom a five-year audiogram had been charted comprised 44 per cent of the total. (2) Twenty-six per cent (52 patients) had returned questionnaires but had not been tested audiometrically at the end of the five-year period. (3) Patients who had neither returned a questionnaire nor been tested audiometrically numbered 60 and comprised 30 per cent of the total number.

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I should like to emphasize that so small a series as 88 cases does not lend itself to statistical analysis since the variables are many and statistics such as these at best may be misleading.

The variables, occurring over the five-year period under consideration, which may have introduced factors of error into our analysis are changes in the state of health of the patient, alterations and changes in the testing equipment used, substitution of audiometric technicians, and changes in the circumambient noise levels under which the audiometric tests were done. Our analysis of operative results under these circumstances can at best but indicate a trend. It is with this realization that I am making my report a matter of record.

The trends or tendencies which seem to be established by study of our postoperative results in both the group reported on previously, which was studied two years after performing fenestration surgery, and the group of 88 patients who were studied five years or more after performance of fenestration are as follows:

- (1) Fifty-five per cent of all patients operated on have less than a 30 decibel loss two years following the operation.
- (2) Thirty-four per cent of all patients operated on have less than a 30 decibel loss five years following fenestration. This would suggest that about one-fifth had dropped below the 30 decibel level between the second and fifth years following fenestration.
- (3) Sixty-five per cent of the patients are pleased with their hearing result, according to the questionnaire, and 56 per cent do not feel the need of a hearing aid five years after the operation.
- (4) All other factors being equal, the better the nerve function before surgery, the better the end result appears to have been.
- (5) If the hearing gain in the "ideal" case drops two years after surgery, the degree of loss occasioned by this drop is greater than that occurring in the "borderline" patient under similar circumstances.
- (6) Deterioration of the nerve function seems to occur postoperatively more frequently in the "upper borderline" classification of patients. This might indicate that this group is in a transitory stage.
- (7) The better the final hearing result from fenestration, the less the tendency toward deterioration of nerve function with passing time.
- (8) Deterioration of nerve function seems to occur a little less frequently in the fenestrated ear than in the opposite unoperated ear. In my opinion, the two greatest causes of failure in obtaining a permanent hearing gain are the improper se-

lection of cases for surgery and the predestined progressive deterioration of nerve function encountered in otosclerosis.

Analysis of our five-year results is gratifying, however, when we realize that little or nothing could be done to improve the hearing for these patients before the development of the procedure of fenestration. In the analysis of our results two years after fenestration, all cases having an average loss of 30 decibels or more in the three speech frequencies were classified as failures. This was true regardless of the decibel gain obtained or the patients' statements regarding their hearing after operation. This same criterion will be used in the report regarding the 88 patients operated on five years or more ago since we were not using speech tests at that time.

I have divided the group operated on two or more years previously and the group of 88 patients who had been operated on five or more years ago into subgroups of "practical" or "nonpractical" hearing according to whether they expressed the opinion on the questionnaire that their hearing was practical or whether audiometric testing showed their hearing to be at the "practical" level according to the criterion mentioned in the previous paragraph.

Of the group of 1,056 who had been operated on two or more years ago, 564, or 54 per cent, had "practical" hearing according to both audiogram and questionnaire. Of the group of 88 individuals operated on five or more years previously, 30 patients, or 34 per cent, had "practical" hearing both by questionnaire and audiogram. This seems to indicate that about one-fifth dropped below the 30 decibel level in the three speech frequencies between the second and fifth year following fenestration.

Of the group of 1,056 who had been operated on two or more years previously, 13 patients, or 1 per cent, were found to have "practical" hearing by audiometric measurement but did not feel that their hearing had been improved to this level according to our subjective criterion, the questionnaire. There was none of this subgroup in the 88 patients operated on five years or more ago.

Of the 1,056 patients operated on two years previously, there were 158, or 15 per cent, who did not meet the established criterion of practical hearing when tested on the audiometer but who, according to the questionnaire, felt that their hearing was at this level. Of

TABLE I
END RESULTS AFTER FIVE YEARS FOR 88 PATIENTS
COMPRISING THIS STUDY

	NO. OF CASES	PERCENT- AGE
Practical on both audiogram and questionnaire	30	34
Improved but not practical on audiogram although practical on questionnaire	27	31
Improved but not practical either by audiogram or by questionnaire	14	16
Unchanged	12	13
Worse	5	6

TABLE II
SUBJECTIVE END RESULTS AFTER FIVE YEARS
FOR 52 PATIENTS WHO RETURNED QUES-
TIONNAIRE BUT FOR WHOM NO AUDIOGRAM
WAS AVAILABLE.

	NO. OF CASES	PERCENT- AGE
Practical	34	67
Improved, but not practical	6	11
Unchanged	6	11
Worse	6	11

the 88 patients who had been operated on five or more years previously, 27, or 31 per cent, fell into this subgroup.

Of the 1,056 patients operated on two years previously there were 321, or 30 per cent, who did not achieve practical hearing either by audiometry or by subjective evaluation. Of the 88 patients operated on five years or more ago there were 31, or 35 per cent, who fell into this subgroup.

Sixty-five per cent of the patients in this study were found to be pleased with their hearing result five years after surgery (Table I). This figure is confirmed by 52 patients who returned the ques-

TABLE III
DROP IN DECIBELS AFTER TWO YEARS BASED ON CLASSIFICATION
(FIVE-YEAR GROUP)

CLASSIFICATION	NO. OF CASES	OPERATED EAR		UNOPERATED EAR	
		AVERAGE AIR DROP	AVERAGE BONE DROP	AVERAGE AIR DROP	AVERAGE BONE DROP
Ideal	3 or 13%	27	19	0	0
Upper Borderline	14 or 61%	11	14	2	7
Lower Borderline	3 or 13%	6	13	0	10
Not Suitable	3 or 13%	13	12	8	10

TABLE IV
AVERAGE NERVE DETERIORATION IN DECIBELS IN OPERATED AND
UNOPERATED EAR FIVE YEARS POSTOPERATIVE.

Classification	No. of Cases	OPERATED EAR				UNOPERATED EAR			
		Preoperative Air	Preoperative Bone	Postoperative Air	Postoperative Bone	Preoperative Air	Preoperative Bone	Postoperative Air	Postoperative Bone
Practical Cases	30	50	8	25	5	40	10	48	17
Improved Cases	27	58	13	38	17	53	13	58	20
Unimproved Cases	31	55	13	58	28	48	18	53	23

tionnaire but upon whom audiometric studies were not available at five years (Table II).

The greatest percentage of drops after two years occurred in the upper borderline group (61 per cent) (Table III). This may indicate that the upper borderline patient is in a transitory stage, whereas the ideal case has not entered that cycle, and the lower borderline and non-suitable groups have already undergone most of their deterioration. These figures also indicate that the ideal case, when and if it does drop, drops to a greater degree than any of the remaining three categories. The average loss by bone conduction

somewhat parallels the average loss by air conduction in the operated ear. In the ideal operated case, it appears that the degree of deterioration is not shared in the opposite unoperated ear. In the remaining three categories, the deterioration appears to be more equally shared in the unoperated and operated ear.

Table IV, which compares the ears receiving surgery with those not operated upon, would seem to indicate that the degree of deterioration of nerve function is less in the ear receiving surgery when the case is successful, but that this does not hold true in the less successful cases. The degree of nerve deterioration seems to be a little greater in the unoperated ear than in the ear that has retained practical hearing five years after surgery. The ear that has been improved by surgery but has not retained practical hearing over a five-year period seems to show about the same degree of nerve deterioration as the opposite ear which has not been operated upon.

Over the five-year period bone conduction in 27, or 31 per cent, of the 88 cases subjected to surgery dropped over ten decibels, while 29, or 44 per cent, of the 66 control ears showed the same degree of drop. (The difference between the number of ears undergoing surgery and the number of control ears is due to the fact that a number of patients received surgery on both ears.) This substantiates the opinion of some that the hearing by bone conduction in the unoperated ear tends to drop in a greater percentage of cases than it does in the operated ear.

Three cases dropped suddenly both by air and by bone conduction two years or more after surgery.

REPORT OF CASES

CASE 1.—This patient developed an upper respiratory infection four years after fenestration surgery. Immediately thereafter her hearing dropped 27 decibels by air and 15 decibels by bone conduction. The fistula test remained strongly positive and there was no apparent clinical cause for her hearing loss.

CASE 2.—This patient developed a sudden loss of hearing in the fenestrated ear of 25 decibels by air conduction and 19 decibels by bone conduction. The loss was not accompanied by vertigo or any other symptomatology. The loss is unexplained.

CASE 3.—This patient developed severe vertigo which persisted for three days, following which there was a loss by air conduction of 60 decibels and by bone conduction of 33 decibels. This loss was unquestionably vascular in origin.

TABLE V
VERTIGO FIVE YEARS POSTOPERATIVELY IN 88 CASES OF
FENESTRATION SURGERY.

DEGREE	PRACTICAL		IMPROVED		UNIMPROVED	
	NO. OF CASES	PERCENT- AGE	NO. OF CASES	PERCENT- AGE	NO. OF CASES	PERCENT- AGE
None	19	63	13	48	15	48
Slight	11	37	12	44	9	29
Marked	0	0	2	8	7	23
Totals	30		27		31	

The average postoperative drop in hearing of patients from 30 to 40 years of age was 23 decibels in air conduction and 20 decibels in bone conduction. For the age group from 40 to 50 years the drop in air and bone conduction was 15 decibels and 18 decibels respectively. For patients whose ages were between 50 and 56 years the drop in air conduction was 11 decibels and in bone conduction, 12 decibels. The younger the patient the greater the apparent drop in hearing. This younger age group probably represents the more ideal case and corresponds to our findings presented elsewhere in this paper.

There were no cases of marked vertigo in the practical hearing group (Table V). Eight per cent of the improved cases noticed marked vertigo, whereas in the unimproved cases 23 per cent were disturbed by this symptom. It would appear that the better the hearing result, the less the vertigo.

All cases in this study had had a dry cavity at some time during the five-year period. There was no discharge at the time of inquiry in 75 cases (85 per cent). The five-year questionnaire revealed that 14 per cent were having slight discharge from the ear at the particular time they received the inquiry, while in one case the discharge was marked.

All patients prior to the fenestration operation had lost serviceable hearing and should have been wearing a hearing aid. Five years after fenestration surgery, 27 per cent of the patients were wearing

a hearing aid and an additional 17 per cent stated that they felt the need of a hearing aid. This represents a total of 44 per cent of the patients under study who are either wearing or who feel the need of an aid. Conversely, 56 per cent of the patients upon whom the fenestration operation was performed are getting along satisfactorily without a hearing aid five years after surgery.

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DISCUSSION

DR. JULIUS LEMPERT: Mr. Chairman, Ladies and Gentlemen, I don't see any room for discussion of a report like this. Those are the results obtained. Here they are. There is nothing to discuss about them.

I may add only one thing, that if I were to analyze the results obtained in a large series of postfenestrated ears I would place least credence or no credence at all to written statements given through the mail in response to a questionnaire sent out to these patients. It has been my experience that a hard of hearing person never knows how hard of hearing he is and when he does know he hates to admit it either verbally or in writing. There is only one way to determine your end results following the fenestration operation and that is by examining the patients audiometrically and comparing your audiometric tests obtained prior to operation, with your various tests made since the operation including the final test made immediately prior to your reporting. I found that all other means of evaluating an end result following the fenestration operation are absolutely worthless.

It is not unusual for some postfenestrated patients, when making an appearance several years postoperative to my institute, either voluntarily or at my request, for a periodic checkup of their ears I would proclaim how wonderfully well they hear, despite the fact that in conversing with them it becomes quite obvious to me that their hearing is no longer practical. Audiometric testing of the operated ear in such patients usually corroborated my clinical observation of a recession in their postfenestrated hearing improvement and rarely if ever substantiated their boastful expression of good hearing.

That is my experience with hard of hearing people. Just as it is true that many deafened people refuse to wear a hearing aid because they believe their hearing is not bad enough to warrant the need of a hearing aid, so it is true that some postfenestrated patients who had their hearing restored to the practical level, and subsequently lost this hearing improvement, believe that they hear well and refuse to wear a hearing aid. But when you test them audiometrically that tells you the story, whether their practical hearing improvement was continuously maintained or not.

Having accepted his preoperative audiograms as an index of the degree of his hearing loss necessitating fenestration for the improvement thereof, I see no reason why a postoperative audiogram showing improvement, no improvement or further loss should be questioned or doubted. A postfenestrated patient's state-

ment of satisfaction with his hearing result in response to a questionnaire is scientifically not acceptable proof of good hearing unless his statement can be corroborated audiometrically. He may be satisfied. Ask his employer whether he is satisfied with him. Ask his sister, ask his brother, ask her husband, and they can best judge. Statistics of hearing results following fenestration are worthless unless these are based upon postoperative audiograms obtained within a reasonably short period prior to reporting.

All these questionnaires are meaningless because hard of hearing people hesitate to place themselves on record in writing as being unable to hear for fear that at some future date those records may be used against them. They are people who want to be employed. These people are constantly trying to convince everybody that they hear well. Unfortunately the hard of hearing can often hide their infirmity. That is one of the reasons that these people are so difficult to rehabilitate unless they are mentally prepared and willing to tell the world that they are hard of hearing.

That is the reason I am always averse to the less and less visible hearing aids which the manufacturers are trying to make all the time. An invisible hearing aid does not rehabilitate the deafened person. He must wear a visible hearing aid so he cannot deny his deafness. The blind comparatively lead a much happier existence than the deafened since their infirmity cannot be denied or hidden and is obvious to every one they come in contact with. If a deafened person would wear a piece of string suspended from one of his ear lobes everybody would immediately realize that he is hard of hearing and talk louder in his presence and he would not even need an electric hearing aid.

Since it is a universally acknowledged fact of life that the hard of hearing people, whether for social or economic reasons of their own, do not like to admit their infirmity, a carefully taken audiometric reading of their function for pure tone sound is to date the most scientific means of determining the degree of a hearing loss prior to the fenestration operation and the degree of hearing gained following fenestration. It is my belief therefore that a postfenestrated patient's verbal or written testimonial or statement of satisfaction with his hearing improvement when not corroborated audiometrically remains doubtful as to the result obtained and therefore is not reportable.

Thank you!

PRESIDENT DAY: This paper is now open for further discussion.

Dr. Macfarlan!

DR. DOUGLAS MACFARLAN: I must apologize for coming up and speaking on the subject because I am more or less an outsider on the fenestration operation, except inasmuch as I do a considerable amount of testing on hearing in deafness cases of all types. I make it a point in my practice to follow my cases carefully year by year, for many reasons which are obvious, and among the things that I found, particularly with the unoperable otosclerotic, is in following them up as they get older, I have come to believe that after about the age of 65, the otosclerotic process becomes more or less arrested. I think this perhaps explains the slide that showed that in those older cases, there was less progression of the deafness from otosclerosis which appeared in the younger groups to be more rapidly progressive.

PRESIDENT DAY: Is there any further discussion of Dr. House's paper? Dr. Walsh!

DR. THEODORE E. WALSH: Mr. President, I want to congratulate Dr. House on a honest and straightforward report.

I cannot agree altogether with what Dr. Lempert has said because some of the patients will give you a reliable answer to a questionnaire. I don't think questionnaires are quite as believable as the pure tone audiometric tests. However, I am not sure that pure tone audiometric tests give you the answer either. Many of these patients, as Dr. House has said, have had an initially good result and then dropped a few decibels. I believe they are just as well rehabilitated as it is possible to get them.

I don't think the criterion of the 30 decibel loss by pure tones will stand up. There is one way to test patients and that is by spoken word to get the social adequacy index. As soon as we get that, we will get an idea of what patients hear.

I was interested in another point that Dr. House made about the bone conduction series. At first it seemed to me that the fenestration operation apparently had not had any effect on stopping further nerve degeneration, and yet later on it seemed as though it had. I wish he would clear that point for me a bit. I must say I am confused. Personally, I have not seen any great change in the discrimination scores in patients in spite of the fact that we have, just as he has, had a great many of the patients drop some decibels at threshold after a period of time. It has been a common finding. As a matter of fact, we have had maximum improvement and then after a year or two a few decibels' drop at threshold but it does not mean that the patients are not rehabilitated.

DR. LEMPERT: I would like to call attention to one thing, to what Dr. Theodore Walsh just said, which is, that the audiogram is not a true estimation of a patient's hearing ability when compared with speech tests. I would like to call his attention to the statement made by Dr. Davis that in testing Dr. Walsh's patients with the social adequacy tests and comparing the results with the tests made on the same patients with audiograms for pure tone, he was not able to find any appreciable difference in the results obtained. Am I right?

DR. WALSH: Not quite. The thresholds compared favorably but that does not mean that the social adequacy index does. It is quite different.

DR. LEMPERT: Breakey and Davis stated in the March, 1949, issue of the Laryngoscope as follows: "Evidently the binaural threshold for speech intelligibility behaves very similarly to the binaural threshold for pure tones and the binaural advantage is approximately 3 db." Maybe I am wrong in my interpretation of their statement. However, experience has taught me that a carefully taken audiometric reading preoperatively or postoperatively for air-borne sound in the presence of good bone conducted sound is the truest estimation of a patient's hearing ability. Speech testing in the language the patient is most familiar with does not necessarily convey the hearing ability of the tested ear. Response to speech testing of this type is more likely to be the result of the patient's ability to interpret the spoken word which ability does not necessarily represent hearing function alone.

PRESIDENT DAY: Are there any other discussers? If not, I am going to interject a comment. To me this is a most gratifying and enlightening paper.

The first thing it pointed out to me is the truth of the fact that this is purely a mechanical procedure when you unlock the labyrinth, that this procedure does

not have any effect on continued bony changes which may cause further deterioration of hearing, and we do get these changes.

I personally, as far as questionnaires are concerned, believe what the patient tells me means a lot. As far as pure tone audiometry, I have seen variations of pure tone audiometry which would make our other tests ridiculous. Accuracy of pure tone audiometry depends upon who does the testing and the conditions under which the tests are made.

I will ask Dr. House to close the discussion.

DR. HOWARD P. HOUSE: Audiometrically, after five years 34 per cent of our cases have less than a 30 db. loss in the three speech frequencies. This means one-third of the fenestration patients at five years are above the 30 db. level.

I certainly agree one can do most anything with a statistical analysis. Even though questionnaires are of doubtful value, I get more gratification when patients state by questionnaire they are satisfied with their surgery, than when the questionnaire is returned with the statement that the result is not satisfactory.

Unfortunately, we were not using speech testing five years ago and, therefore I have not reported speech findings after five years. As Dr. Walsh knows, we are now using speech and have been for the past three years. I believe the answer to this argument of audiograms vs. speech testing is simply the more the merrier. Therefore, the more information we can obtain with questionnaires, audiograms, and speech testing, the more accurate will be our final analysis.

I would like to thank my discussers for their contributions to this paper.

PRESIDENT DAY: We will hear the "X-ray Visualization of the Attic-Aditus-Antral Area of the Chronic Mastoid Kodachrome with Narration" by Dr. Gilbert Roy Owen.

Abstracts of Current Articles

EAR

Observations About the Presumed Action of Sodium Bicarbonate on the Otolithic Crystals.

Giaccai, Fabio, and Carle, Aldo: Arch. Otolaryng. 53:434-438 (Apr.) 1951.

The authors report that Hasegawa stated abnormal stimulus of the otolithic apparatus expressing itself thru the vagosympathetic system is responsible chiefly for motion sickness. Motion sickness is banished in animals whose otolithic crystals have been destroyed by centrifugation or animals on which sympathectomy has been performed and animals deprived of their otoliths do not exhibit disturbances of balance. Therefore, Hasegawa raised the question of the advisability of destroying the otoliths in man to prevent motion sickness. He sought a method practicable to use in man and stated that he found sodium bicarbonate injected intravenously to be the most suitable agent and confirmed this by work on rabbits and man. After treating the subjects according to his technic he noted, on histologic examination, that otolithic crystals were absent; the subjects, when put in situations conducive to seasickness, did not become seasick following treatment. Hasegawa offered as his hypothesis for the mechanism operating in the destruction of the otoliths by the sodium bicarbonate that this occurs when carbon dioxide generated under the otolithic apparatus transforms the otolithic crystals of calcium carbonate into a soluble calcium bicarbonate. He based this on his observation that if carbon dioxide were bubbled into a test tube containing otolithic crystals in aqueous suspension, their presence could not be confirmed by microscopic examination.

The authors wished to assure themselves that sodium bicarbonate did have an action on the otolithic apparatus. They point out the fallacy of some of Hasegawa's observations in that they were on decalcified and then sectioned labyrinths in which they felt the decalcification process could have dissolved the otolithic crystals. In vitro they could observe otolithic crystals microscopically 24-48 hours after placing them in five and nine per cent solutions of sodium bicarbonate; they obtained uncertain results by bubbling carbon dioxide using Hasegawa's technic. They, therefore, treated a series of pigeons with intravenous injections of different concentrations of a solution of sodium bicarbonate (one series received repeated injections) and observed histologically whether the otolithic crystals

were destroyed. In every instance the otolithic crystals were still present and morphologically similar to those of normal control pigeons.

To determine if sodium bicarbonate had any effect on the function of the otolithic crystals by checking the reflexes originated in them, a series of guinea pigs was subjected to injection of various concentrations of sodium bicarbonate into the jugular vein under carefully controlled conditions. The reflexes of the treated animals were perfectly normal and they behaved exactly like the normal one and very differently from one in which the labyrinth had been destroyed.

These experiments, the authors conclude, exclude the possibility that sodium bicarbonate has any action on the otoliths or otolithic reflexes.

HILDING.

Is Hearing Loss Due to Nutritional Deficiency? Further Studies on the Influence of Vitamin A to Certain Types of Impaired Hearing.

Lobel, M. Joseph: *Arch. Otolaryng.* 53:515-526 (May) 1951.

Clarification of the possible role of vitamins in deafness and the presentation of results of more extended studies with an improved injectable vitamin A product led the author to follow his earlier paper on "Clinical Studies with Parental Vitamin A Therapy in Deafness" with the present report.

In an extensive review of the literature he brings out the controversial theories held on the relation between vitamins and deafness but concludes that despite adverse opinions there is substantial foundation for the belief that there is a relationship between vitamins and deafness.

In the author's experiments he selected vitamin A for the treatment of deafness for a number of reasons which he supports with references to the literature. Six patients, with varying degrees of hearing loss, were treated primarily with Vitamin A and their case histories, including audiographic tracings before and after treatment, are presented.

In commenting on the studies which have been made, the author points out the necessity for considering the patient's general physical condition and suggests laboratory data which may prove helpful to the otologist in such entities as Bright's disease and cirrhosis of the

liver, for example, which have been studied by him because of the incidence of associated tinnitus and loss of hearing.

Lobel concludes that his clinical study seems to confirm the views held by some workers that the results of clinical trials with a new injectable vitamin A preparation (Anatola) hold out promise of its therapeutic value in certain types of deafness. In some instances hearing loss was not influenced but the accompanying tinnitus was markedly ameliorated. The results of studies to date seem to him to justify further trials with vitamin A, alone or combined with other vitamins, in the treatment of hearing loss.

HILDING.

Observations of the Eustachian Tube.

Perlman, Henry B.: Arch. Otolaryng. 53:370-385 (Apr.) 1951.

The author feels that knowledge of the physiologic role of the middle ear as a ventilator is limited and therefore additional experimental observations are necessary, but these are technically difficult to devise. A brief review of the scant literature on the subject is given.

An experimental method for obtaining objective quantitative data on some acoustic phenomena related to tubal function is described and studies are recorded on autophony and acoustic transients. In the former, he determined the acoustic nature and degree of sound pressure changes present and he recorded the latter in association with swallowing and voluntary opening of the tube. He found that palate and tubal activity produced in phonation was not associated with opening of the tube. In opening the tube, swallowing water was more effective than swallowing without water, and he observed that the tube was frequently closed even during swallowing. The reflex opening of the tube was different from the voluntary opening in degree, duration and rate of opening and closing.

Dr. Perlman points out the fact that the eustachian tube and middle ear are physiologically exposed to sound pressures in the nasopharynx during phonation and also to slower pressures incident to respiration and deglutition. The work of Bekesy, elucidating the nature of sound transmission thru the head during phonation, is discussed in some detail. Observations on Toynbee's maneuver are made and a possible explanation therefor offered.

HILDING.

Mouth of the Eustachian Tube: Action During Swallowing and Phonation.

Perlman, Henry B.: Arch. Otolaryng. 53:353-369 (Apr.) 1951.

Conflicting ideas and studies, from the middle of the 19th century to the present time, in man and animals, on the action of the muscles in the region of the eustachian tube are described.

The author had four patients with facial defects which permitted observation directly from above and motion pictures of the tubal area were made with a magnifying lens at from 16 to 64 frames/sec., recording frame by frame the motion sequences associated with phonation and swallowing, thus enabling the observer to more easily reconstruct and time the details of the movement of the soft palate and tube during phonation and swallowing than with unaided vision. His observations of the movements filmed on phonation and swallowing are minutely described. In addition, he states that to determine whether the resting tube is normally open or closed and to determine the changes in the continuity of the air molecules in the middle ear with those at the mouth of the tube during physiologic action, other methods than direct inspection of the mouth of the tube must be employed. Manipulations of the ears of his subjects did not result in reflex activity of the tubal muscles. Moderate pressure applied on the neck produced narrowing of the tubal lumen.

HILDING.

Prophylactic and Therapeutic Control of Vestibular Disturbances with Dimenhydrinate.

Gay, L. N.: J. A. M. A. 145:712-715 (Mar. 10) 1951.

The author discusses the investigations which have been made upon the action of dimenhydrinate (dramamine), one pointing to a central action and others to local action on blood vessels of the labyrinth. He refers to the report of Campbell indicating the efficacy of dimenhydrinate in labyrinthine disturbance after fenestration. He concludes that the benefits that the patients derived from the drug show that dimenhydrinate should be given to all persons with disturbance of balance before recourse to section of the nerve by the neurosurgeon or to destruction of the labyrinth with alcohol injection or to electrocoagulation by the otologist.

BOIES.

Effect of Streptomycin on Vestibular Function.

Bignall, J. R., Crofton, J. W., Thomas, J. A. B.: *British Medical Journal*, No. 4706 (Mar. 17) 1951.

The effects of streptomycin on the vestibular apparatus were investigated in 76 cases under treatment for pulmonary tuberculosis.

Patients receiving 2 gms of streptomycin a day complained of giddiness almost four times as often as those receiving 1 gm and the giddiness was first noted significantly earlier.

Nystagmus and abnormalities in the caloric tests were often associated with a complaint of giddiness. Clinical tests for coordination of movement, such as walking along a line with the eyes shut, were useful only when there was gross ataxia; minor degrees of ataxia were difficult to interpret in patients who had been confined to bed, and some cases with absent caloric responses had normal walking tests.

An attempt was made to prevent vestibular damage by giving antihistamine drugs prophylactically to patients receiving streptomycin. The results suggest that some success was achieved, but the evidence was not conclusive.

BOIES.

NOSE**Distribution and Comparison of Nasal Cilia.**

Tremble, G. Edward: *Arch. Otolaryng.* 53:483-491 (May) 1951.

The author briefly sketches the development of the knowledge of cilia and ciliary action since cilia were first noted in 1683. He traces the existence and action of cilia in various forms of lower animal life and correlates that with what is known of human cilia and ciliary activity.

Tremble reiterates the well known fact that the exact mechanism of ciliary movement is uncertain and then lists the following facts which have been established:

The motility of a cilium does not depend on the cell structure as a whole to which it is attached. It is generally agreed that the motive stimulation of the cilia lies in the distal portion of the cell, i.e. just below the surface.

In the human embryo, the nasal cilia develop early from three germinal layers—ectoderm, mesoderm and entoderm, and appear at

the fourth fetal month. Shortly after birth the nasal cilia seem identical microscopically with adult cilia. The frequency and amplitude of their beat are similar at various age periods. Human cilia are resistant and powerful structures and often continue beating many hours after death.

One investigator reported that active movement almost always exists within a sinus, even in the presence of severe infection. After removal of the mucous membrane lining of a sinus, regeneration occurs in about five months with normal appearing mucous membrane with active cilia.

The human cilia are 5-7 microns in length, varying slightly in different areas; they are just under 0.3 micron in diameter and the distance between the cilia is about 0.3 micron. There are probably 20-30 cilia to a cell and 250-300 complete cycles/min.

Except for a few areas (the anterior third of the septum and lateral wall, the olfactory area, pharynx and edges of the vocal cords in an adult), the whole respiratory tract is lined with ciliated epithelium. Histologic sections illustrate the high columnar surface cells and relatively short cilia in the middle third of an inferior turbinate and the short columnar cells and long, well developed cilia in a middle meatus. The nasal mucous membrane varies in thickness, reaching a maximum in areas most exposed to outside air with its associated contaminants, and becoming progressively thinner as the lesser exposed areas are reached until, in the sinuses, it is thinnest of all. The depth of the stroma varies with the depth of the epithelial layer, being widest in the exposed anterior portion of the nose and disappearing almost entirely in the sinuses.

The work of various investigators is described and reference made particularly to a comprehensive article by Ballenger on ciliary activity.

HILDING.

Nasopharyngeal Angiofibroma in a Girl.

Parchet, Victor: Les Annales d'Oto-Laryngologie 68:60-71 (Jan.) 1951.

The author reports a case of what appeared to be a juvenile nasopharyngeal fibroma, occurring in an adolescent girl. The case is reported because of the unusual occurrence of this tumor in a female. The history of the case, the gross appearance and location of the tumor and its histological characteristics were all those of the usual juvenile nasopharyngeal fibroma occurring in adolescent males. The

tumor was removed through the mouth, after retracting the soft palate. There was profuse bleeding, which was controlled by tamponade.

A review of the literature revealed only two cases of juvenile nasopharyngeal fibroma, demonstrated histologically, to have been reported as occurring in adolescent females.

HARKINS.

Treatment of Adenoid Remnants by Radium (Method of Crowe-Burnam).

Moulinard, J.: *Les Annales d'Oto-Laryngologie* 68:73-86 (Jan.) 1951.

The author has demonstrated that the energy emitted from the Crowe-Burnam radium applicator is 93% beta radiation and 3% gamma radiation. He points out that it is the amount of energy absorbed in the tissue that constitutes the dose. One minute of application corresponds to 370 physical equivalent roentgens. All of this beta radiation from the Crowe-Burnam applicator is absorbed in or very near the surface; in other words, the penetrating power is slight: 85% of the energy is absorbed in the first three millimeters of tissue. He also shows that in the relatively short time the applicator is used, lengthening the time of exposure (for example, from five to 20 minutes) does not appreciably increase the resulting radio-epithelitis. But he states that 20 minutes interval of time is the upper limit of safety for exposure of the patient. One could not expect to obtain much radiation in depth, from the penetrating gamma rays, in so short a time, since the gamma radiation is such a small part of the total quantity of the rays. To obtain adequate, penetrating gamma rays would require radium shielded in platinum over a period of many hours, a procedure which would not be practical, particularly since the majority of these cases are in children.

The Crowe-Burnam applicator has its place, according to this author, in the treatment of adenoid remnants in the nasopharynx, in the treatment of lymphoid tissue lying immediately below the surface, around the pharyngeal orifice of the Eustachian tube, not exceeding a depth of three millimeters. It can only supplement x-ray therapy, which is able to reach lymphoid tissue lying deeper, along the walls of the Eustachian tube, throughout its entire length. While there is no danger for the patient, when the Crowe-Burnam applicator is used properly, the repeated use of the applicator is a very real danger to the attendant and his assistants.

HARKINS.

Tuberculosis of the Nasolacrimal Duct.

Konstam, P. G., and Meynell, M. J.: *The Lancet* 260:443, No. 6652 (Feb. 24) 1951.

A case is reported of what was apparently a primary tuberculous complex in the nasolacrimal duct. The case was in a child of 14 months. Preauricular and submandibular glands were involved and partial dacryocystectomy was done. Presumably the original source of infection was in the father, who had a history of phthisis and whose sputum had contained tubercle bacilli. Both the mother and the child were infected. A course of streptomycin giving 0.25 gm six-hourly was instituted. However, about two weeks later a fluctuant swelling over the submandibular region developed and was aspirated. The child was then given "Calciferol" in the dosage of 50,000 units daily in addition to the streptomycin. Despite this therapy the swelling increased so it was excised about one month after the streptomycin had been started. The entire mass of superficial and upper deep cervical glands was removed. These were found to have been broken down and were in different stages of destruction. Caseation was present. A total of 31.5 gms of streptomycin was given over a period of four weeks and two days, and 800,000 units of "Calciferol" in 16 days. The lesions dried and began to heal with some scarring. A further flareup of the lesion, however, occurred over the nasolacrimal duct and partial dacryocystectomy was done. Healing again occurred and the patient had remained well up to the time this report was made, which apparently was about a year later.

BOIES.

THROAT**Relation Between Tonsil and Adenoid Operations and Cleft Palate.**

Beatty, H. G.: *J. A. M. A.* 145:379-382 (Feb. 10) 1951.

The author describes a method of tonsillectomy in cases of cleft palate which he calls tonsillectomy with extension of the palate. The operation was originated by Hudson-Macuen and modified by Brophy. The author believes that no child should be denied the removal of obstructive or infected pharyngeal lymphoid tissues. One should be concerned, however, about the possible effects of this surgery on the functioning of the soft palate and the production of speech. From the method he describes he believes that the necessity for any more formidable surgical procedures involving the palatal structures may be avoided, such as the "push-back" operation.

Operation is preferably delayed until the child is six or seven years old, but this is not always possible. He removes all the tonsil in some instances; in others he leaves a strip of tonsil and capsule at its mesial attachment of the palatopharyngeus muscle. The uvula is partially removed, and the borders of the palatopharyngi muscles are carefully split as far ventrally as is desired for union, and usually a little farther, as over-correction is a good procedure for compensation for contracture and some possible separation. The dorsal borders of the muscles are then united with fine horsehair sutures and knots tied on the nasopharyngeal surface. The anterior borders are then closed with fine horsehair. As the closure approaches the lower or ventral extent, the muscle is cut through sufficiently in a tranverse direction for the attainment of relaxation. This cutting of the mesial portion of this muscle does not noticeably impair the function of the new palate, because the innervation of the muscle is not damaged by a partial section. Bleeding is controlled. Silver wires are introduced through the palatoglossi and newly closed palatopharyngi muscles which are to be united over lead plates as in the soft palate cleft closure. A rubber breathing tube is passed through one nostril down to the lower border of the closure. This facilitates easy breathing and lessens respiratory vibration of the new structure. The tube is removed in 24 hours. Again the tension of the wires on the lead plates must be correct and careful daily attention is necessary during the following days, as the approximated flaps are not a sturdy structure like the velum. This results in a good extension and gives satisfactory functional results. If the tonsils are small and the author believes that there will not be too much contraction, this extension is not done.

BOIES.

Cortisone in the Treatment of the Poststreptococcal State of Hemolytic Streptococcal Sore Throat.

Doerner, A. A., Naegle, C. F., and Regan, F. D.: J. A. M. A. 146:641-643 (June 16) 1951.

A case is presented of a young white woman who had a typical hemolytic streptococcal infection of the throat. She received early and adequate antibiotic therapy, but continued to manifest prolonged unremitting fever and had subsequent complications of a scarlatiniform rash, transient arthritis of the knee and ankle, a mitral systolic murmur, a transient pericardial friction rub, a left lower lobe pneumonia and pleural effusion, anemia, leukocytosis with pronounced neutrophilia, progressive generalized toxicity, and a downhill course leading to extreme disability.

These complicating nonsuppurative sequelae of hemolytic streptococcal disease can be referred to as the poststreptococcal state. It has been suggested that this state occurs not as the result of the primary etiological agent per se, but as a more complex mechanism by which the patients' tissues have been sensitized to some fraction or product of the hemolytic streptococcus, probably as a result of previous exposure to this organism.

This case is presented in considerable detail, with charts illustrating the clinical and laboratory course of this patient, and her complete lack of response to specific antibiotics, salicylates and general supportive measures.

The patient was treated with cortisone and there followed immediately a rapid disappearance of all the features of the poststreptococcal state. The authors comment upon the mechanism of this favorable influence and on the agreement with Hensch who observed that the action of cortisone appears to be group specific rather than disease specific or nonspecific, and that cortisone perhaps constitutes one of the body's chief defenses against the action of certain agents on certain tissues.

BOIES.

"Sore Throat" in General Practice.

Landsman, J. B., Grist, N. R., Black, R., McFarlane, D., Blair, W., and Anderson, T.: British Medical Journal, No. 4702 (Feb. 17) 1951.

This is a study on "sore throat" as it is seen in ordinary urban practice. The study is not complete. The results reported cover the first 100 cases. It is a well controlled study. The results of the therapy in their cases do not support the thesis that sulphonamides exert a beneficial effect in the treatment of the "acute throat." The authors remarked that those who place more faith in the virtue of clinical impressions will no doubt be unconvinced by an array of "statistics." The authors contend that in view of their findings, added to the fact that in about half the cases a streptococcal etiology is unlikely, it seems that to use drugs which have definitely known dangers is unnecessary, besides being expensive.

BOIES.

Sulphonamides and Acute Tonsillitis.

MacDonald, T. C., and Watson, I. H.: British Medical Journal, No. 4702 (Feb. 17) 1951.

The authors are attempting to solve a controversy which resulted from the report of Anderson in 1929 to the effect that the

efficacy of a sulfonamide for the treatment of acute sore throat has never been convincingly demonstrated, and because of the fact that sulphonamides are undoubtedly toxic materials carrying appreciable risks of sensitization, their administration is inadvisable, and in certain conditions may even be dangerous.

The authors studied 82 consecutive cases of acute tonsillitis divided into two equal groups in random manner. They were all treated by rest in bed and give copious fluids to drink. One half were given a total of 50 0.5 gram tablets of "sulphatriad." One tablet was given every four hours, except between 10 p.m. and 6 a.m., along with two grams of potassium citrate. Fluids were administered liberally. The other half were given an equal number of lactose tablets, identical in appearance to the "sulphatriad."

Analysis of clinical records revealed no difference between controls and treated cases in the speed of recovery from various symptoms. The medical officer in clinical charge was, however, able to make an estimate of "goodness of cure" which had a significant relationship to the administration of "sulphatriad."

It was felt that the routine administration of sulphonamides to patients with acute tonsillitis cannot be supported by the results of this experiment.

BOIES.

PHARYNX

Live Fish Caught in the Hypopharynx. Extraction by Direct Laryngoscopy.

Tam, P. B., and Ninh, T. N.: Bronchoscopie, Esophagoscopie et Gastroscopie, pages 32-35, 1951.

From Hanoi, French Indo-China, comes this report of a boy who got a small fish in the hypopharynx while fishing in a rice-pool. The boy was holding the live fish between his teeth; the fish gave a sudden lunge and broke loose, slipping back into the throat. The head of the fish was lodged in the upper entrance of the esophagus; the body lay across the upper orifice of the larynx. The tail was uppermost; it was the presenting part and was seized with laryngeal grasping forceps in removing the invader. The fish was identified as *Anabas testudineus*, commonly called "Ca Ro" in Indo-China.

HARKINS.

Nasopharyngeal Irradiation.

Garland, L. H., Hill, H. A., Mottram, M. D., and Sisson, M. A.: J. A. M. A. 146:454-461 (June 2) 1951.

The authors compare the relative merits of roentgen and radium therapy for benign conditions in the nasopharynx. They discuss the anatomic and pathologic considerations and symptomatology, and review some of the literature. Their observations are based upon personal experience in the treatment of 720 cases. Excellent results with roentgen therapy are reported in from 60 to 90 per cent of the cases. These results, they state, were validated by independent clinical observation of the referring physicians, many supplemented by audiometric and other objective tests. They discuss the relative advantages and disadvantages of roentgen and radium therapy and illustrate the distribution of ionization through the involved tissues in the adjacent area. They contend that the dosage throughout the zone commonly involved in lymphoid hyperplasia is more homogenous and efficient with external roentgen beams than with intranasal radium beams. They also contend that the dose in the immediate proximity of the radium applicators is some ten times the dose ordinarily regarded as reasonable or safe for the treatment of non-malignant conditions. The conclusion is that the maximum tissue dose required for cure when roentgen therapy is employed is much lower than when standard radium applicators are used and that roentgen therapy is considerably safer and should, in their opinion, be employed as the method of choice.

BOIES.

LARYNX**Treatment of Tuberculosis of the Larynx by Chemotherapy.**

Wallner, L. J., Turner, G. C., Lichtenstein, M. R., and Sweany, H. C.: J. A. M. A. 145:1252-1254 (Apr. 21) 1951.

The results of treatment are reported of 70 patients with laryngotuberculosis who were given streptomycin and/or para-aminosalicylic acid. Some of the early patients were treated with 2 gms of streptomycin per 24 hours for 120 days. In 79.2 per cent of all the patients treated for any form of tuberculosis those who received this dosage had indications of a toxic effect on the vestibular apparatus. The dose was later reduced to 1 gm and then to 0.5 gm a day for 60 days. The present method of treating is to give the dihydrostreptomycin in a dosage of 0.75 gms a day for 60 days. This smaller dosage has been found effective and rarely causes vestibular symptoms.

Para-aminosalicylic acid was given orally in doses of 12 gms a day for 120 days. Some of the patients given para-aminosalicylic acid had streptomycin previously. Their laryngeal disease relapsed or developed after streptomycin therapy. No local treatment of the larynx was given. All patients were advised to stop smoking and rest the voice completely. It is doubtful if either was done conscientiously enough to be of benefit.

All but two of 39 patients with pain were completely relieved. There was some improvement in the hoarseness in all but six; in 41 it ranged from a marked to a complete return of the normal voice. The disease progressed in three patients and remained unchanged in two others. Improvement was slight in five patients, moderate in 23, and marked to complete healing occurred in 37.

Para-aminosalicylic acid is especially valuable to patients whose organisms have become streptomycin resistant.

The prognosis becomes that of the pulmonary condition alone, rather than the poorer one of pulmonary tuberculosis with laryngeal involvement.

BOIES.

Acute Obstructive Laryngotracheitis and Laryngotracheobronchitis.

Forfar, J. O., Keay, K. R., and Thomson, J.: *The Lancet* 260:181-186, No. 6648 (Jan. 27) 1951.

The authors report a study of an outbreak of 22 cases of acute obstructive laryngotracheitis and laryngotracheobronchitis, which seems to be the first outbreak of its kind in Britain. They discussed the epidemiology, etiology, clinical features, pathology, treatment and reviewed current opinion. They remark that owing to the lack of agreement on what constitutes acute obstructive laryngotracheitis and laryngotracheobronchitis, and to the variation of severity in different outbreaks, no close comparison of percentage mortality figures and methods of treatment between one series of cases and another would be valid. Different methods of treatment are, therefore, difficult to compare. On certain measures, however, they believe that there is general agreement: necessity for a high humidity of the respired air to prevent the secretions from drying and becoming viscid and crusted; the use of oxygen; the giving of adequate fluid; and the contra-indication to atropine and to sedatives which are respiratory depressants.

In the matter of the surgical aspects of treatment, however, they express a very conservative attitude, remarking that in their cases laryngoscopy when done was followed by an increase in the respiratory distress and also that the two cases in which tracheotomy was done, and the one case where laryngeal intubation was done were not improved by this treatment. In most of their cases they relied mainly on the supporting measures referred to above, combined with chemotherapy and antibiotics.

Their over-all mortality in this series was 18 per cent. The mortality among cases classified as severe, of which there were nine, was 44 per cent.

They expressed the belief that chemotherapeutic and antibiotic drugs may, in many instances at least, supplant the surgical measures which in the past have been considered essential in treatment.

BOIES.

TRACHEA

Repair of the Cervical Trachea and Larynx

Lewis, Emlyn: *The Lancet* 260:830-831, No. 6659 (Apr. 14) 1951.

A case is reported of a successful reconstruction of the cervical trachea and larynx in a boy aged 16. The boy had had diphtheria as an infant which had necessitated tracheotomy. The history of the case was not considered to be accurate but it was assumed that secondary infection had occurred at the time of the tracheotomy, and that this infection had destroyed much tissue, the tracheal rings, and part of the thyroid and cricoid cartilages. Many operations had been performed on his neck. Photographs are shown to illustrate the extent of the defect. The details of the construction of the abdominal tubed pedicle flap are shown and the steps by which this flap was utilized are given in detail. The repair proved successful and a year after completion of surgical treatment the boy was working as an electrician. He spoke well with a slightly husky voice. He could exercise without difficulty in breathing. He could expectorate satisfactorily. Photographs illustrate the surgical steps.

BOIES.

ESOPHAGUS

Are Any Strictures of the Esophagus Due to Diphtheria?

Kiviranta, U. K.: *Medicina Fennica* (Finland) 21:81, 1949.

The author describes five cases of esophageal stricture which had been regarded as sequelae of diphtheria, but of which three were definitely found to be due to corrosion (lye) on further examination. In the remaining two cases the diagnosis was based on the patient's own assumptions; yet, the course of the disease in these two cases and the sequelae are indicative of corrosion. The importance of a positive culture of the diphtheria bacillus in cases of corrosion and stricture is discussed. The strictures of the esophagus reported in the literature as "definitely" due to diphtheria are reviewed and the conclusion formed that no single case can be definitely attributed to diphtheria.

HARKINS.

Primary Reticulum Cell Sarcoma of the Esophagus.

Wild, C., Frubling, L., and LeGal, Y.: *Bronchoscopy, Oesophagoscopy et Gastroscopie*, pp. 36-43, 1950.

The authors report two cases of primary reticulum-cell sarcoma of the esophagus. Histological examination revealed the tumor to be composed of a basophilic syncytium, without any evident cell boundaries, and containing numerous round or oval nuclei, which were almost in contact with each other. The nuclei showed the chromatin as dust-like particles. Mitoses were numerous. There was a complete absence of any disposition to form alveoli, and an absence of any stroma. This was interpreted as reproducing a reticulum structure or the fundamental plasmodium of the mesenchyme.

In both cases the duration of the disease was extremely short: in the first case, five months; in the second, three months, from the onset of dysphagia until death. In both cases the tumors attained large size, infiltrating the walls of the esophagus extensively and obstructing the lumen. In the first case intensive x-ray therapy was given (9,000 roentgens) in approximately five weeks, and the tumor is said to have entirely disappeared at the end of this time. These tumors are said to be very radio-sensitive. However, the growth rapidly recurred, a tracheo-esophageal fistula developed and the patient expired. The second patient was not treated. At autopsy extensive metastases were present, in widely scattered lymph nodes throughout the body and in the liver. Both patients were males; one was 61, the other 59 years of age.

HARKINS.

BRONCHI

Bronchoscopy in the Newborn: The Otolaryngologist's Contribution to Obstetrics.

Rosedale, Raymond S.: *Arch. Otolaryng.* 53:393-396 (Apr.) 1951.

Obstetricians have used many indirect methods of restoring respiration to newborn infants but even the often-relied upon aspiration of the trachea with a catheter may result in the catheter's entering the esophagus, where some fluid may be aspirated, yielding false evidence if it is interpreted as coming from the trachea or larynx; laryngeal spasm may be produced by impact with the un-anesthetized larynx or hyperemia and edema of the larynx may be produced. The disadvantages of the indirect methods point the way to the advisability of employing the direct method of bronchoscopic aspiration.

There is a paucity of literature on the subject of bronchoscopic aspiration of the newborn but that which has appeared seems to favor the procedure. One author concluded that bronchoscopy could be done on all newborn babies and many prematures with safety and that greater cooperation between obstetricians, pediatricians and otolaryngologists is necessary for the welfare of the newborn.

The author gives as the purpose of this procedure the resuscitation of those infants whose inability to achieve adequate ventilation is due to laryngotracheobronchial obstruction, whether it be due to physiologic or anatomic abnormality or the presence of an obstructive agent. The procedure serves to remove the obstructive agent, if such is present, as well as to detect possible anomalies. If a diagnosis of aspiration and atelectasis in the newborn cannot be made with certainty, he sees no serious reason for not using bronchoscopy in any questionable case. The oxygen administered with the bronchoscopy makes it a supportive as well as therapeutic measure. In suitable cases it is a lifesaving procedure.

HILDING.

Bronchial Adenoma.

Huizinga, E., and Iwelma, J.: *Les Annales d'Oto-Laryngologie* 67:743-753 (Nov.-Dec.) 1950.

A discussion of bronchial adenomata is given, based on 17 patients with adenomata in the bronchi and three patients with tumors in the trachea (two cylindromata and one adenomata). There was a preponderance of these tumors in females, and in persons under 40

years of age. Characteristically the history in these cases was a long one. Hemoptysis was an important early symptom. Obstruction of the bronchial lumen with resulting atelectasis and bronchiectasis dominated the clinical picture. Bronchoscopy usually revealed a red polypoid tumor, covered with mucous membrane, which had adapted itself to the contours of the bronchial lumen. A biopsy should be taken. While the tumor may be purely intrabronchial, a portion of it may be extrabronchial; yet even this remains encapsulated. The authors have never seen local invasion or metastasis. These adenomata are covered with bronchial epithelium, which often undergoes metaplasia into squamous epithelium. The tumors themselves consist of a small, polygonal or cuboidal cells, having deeply stained nuclei in which there are well defined chromatin granules. The cells are arranged in alveoli and cords, separated by a stroma containing blood vessels. Cell division is scarcely ever seen, and atypical cells are never seen. Often these tumors are very vascular, which explains the hemoptyses and the bleeding which may follow biopsy.

There are several theories as to the origin of this tumor which the authors present and discuss. The authors favor the theory that these tumors develop from sero-mucinous bronchial glands and are comparable to salivary gland tumors. They maintain that this tumor is analogous to adenomata of the salivary glands and, like these, shows an adenomatous form. These authors found that in the trachea and bronchi, cylindromata occur, which are similar to mixed tumors. These cylindromata are not entirely benign; the authors had one case of cylindromata of the trachea in a young man which showed metastasis to the hilar lymph nodes. Also, they have had three cases of adenomata which, when sectioned, showed the structure of cylindromata in certain portions of the tumor. Microphotographs are presented to demonstrate this. The authors state that a similar condition is found in some salivary gland tumors and in tumors of the seromucinous glands of the mouth. The authors state that they have never found any instance, in these bronchial adenomata, of a transition from the bronchial mucosa to tumor cells. In one of their bronchial adenomata they found glandular alveoli, well formed and actively secreting mucous.

Treatment must be fitted to the individual case, considering the extent and location of the tumor, and the age and economic status of the patient. The polypoid type of tumor pushing out into the bronchial lumen is often suitable for ablation through the bronchoscope, using a double curette and electro-coagulation. Cure may result, or the tumor may recur from time to time. Sometimes this

type of treatment is only palliative. Other cases require lobectomy or pneumonectomy, especially if bronchiectasis is present distal to the lesion. Death usually comes, in fatal cases, from chronic sup-puration distal to the growth. An important disadvantage of bron-choscopic treatment is the necessity of following the patient for years for regular bronchoscopic examinations.

HARKINS.

Early Detection of Bronchiogenic Carcinoma.

Paulson, D. L., and Shaw, R. R.: J. A. M. A. 146:525-529 (June 9) 1951.

Observations are reported on 362 patients with bronchiogenic carcinoma seen in a period of three and one half years. In this group pulmonary resection could be applied to only 107, or 30 per cent. The authors stress the point that the over-all salvage rate for bron-chiogenic carcinoma remains disappointingly low in spite of advances in its surgical treatment.

Bronchoscopy is the most valuable of the specific methods of diagnosis. It must be remembered that all parts of the bronchial tree are not visible bronchoscopically. This means that carcinomas arising in the smaller bronchi are not accessible to either visualization or biopsy, but bronchial secretions or bronchial washings may be obtained from the suspected lobe. In view of the limitations of bronchoscopy in the examination of the tracheobronchial tree, it must be remembered that a negative finding in bronchoscopy or failure to find malignancy in either a biopsy or in bronchial secre-tions or washings does not rule out carcinoma. The authors com-ment on lung aspiration or needle biopsy which they regard as haz-ardous and superfluous. They believe that after completion of a careful history and physical examination and a roentgenographic ex-amination of the chest and bronchoscopic examination for an in-determinate lesion in a man over the age of 40, an exploratory thor-acotomy is mandatory and should be accomplished without delay.

BOIES.

Bronchial Involvement in Tuberculosis of Childhood.

Laff, H. I., Hurst, A., and Robinson, A.: J. A. M. A. 146:778-783 (June 30) 1951.

The authors contend that although the recognition of the im-portance of bronchial infections in adult pulmonary tuberculosis has been attained by an increased utilization of bronchoscopy during the past two decades to add immeasurably to our knowledge of this disease, bronchial involvement in childhood pulmonary tuberculosis

has not received the attention that it deserves in America. Observations in 15 cases are reported, and five of these reports are made in detail to illustrate the involvement of the bronchus in different types of primary tuberculosis. These fifteen cases had either wheezing or persistent atypical x-ray shadows. Caseous material was aspirated from seven of them (47 per cent). The point of rupture could not always be visualized at the first bronchoscopic examination, but could usually be demonstrated at subsequent examinations. It is the authors belief that repeated examinations with the bronchoscope will ultimately demonstrate rupture in an increasingly greater percentage of cases studied.

The authors conclude that bronchial involvement in the pathogenesis of parenchymal consolidation and in the course of primary tuberculosis in children is an important and frequently overlooked factor.

BOIES.

Streptomycin Treatment of Tuberculous Lesions of the Trachea and Bronchi.

A Report to the Medical Research Council by the Streptomycin in Tuberculosis Trials Committee: The Lancet 260:253-257, No. 6649 (Feb. 3) 1951.

In six chest hospitals a cooperative trial was made of streptomycin in the treatment of tuberculous tracheobronchitis. Thirty-five patients were treated. The amount of lung disease varied widely; cavitation was present in ten cases.

In 24 (68 per cent) of the 35 patients, the bronchial lesion was found to be greatly improved or healed when the final bronchoscopy was carried out four or six months after the start of treatment. In 15 patients (45 per cent) of 33 who were bronchoscoped two months after starting treatment much improvement in the appearance of the bronchial lesions was noted. As might be expected, healing of the bronchial lesion was more frequent in those with limited lung disease than in those with tuberculous cavities. Some patients received streptomycin in dosage of 2 gm daily, and others 1 gm daily. The authors remarked that the figures are too small to allow a firm conclusion, and there is only a slight suggestion that the lower dosage was less satisfactory. Bronchial stenosis was present before treatment in 15 patients (43 per cent), and in seven more cases it developed during treatment. They remarked that in some cases it may necessitate later surgery. In two cases, in spite of streptomycin treatment, masses of granulation tissue required removal at repeated bronchoscopies.

BOIES.

Books Received

American Laryngological Association. Transactions of the, 1951.

Proceedings of the Annual Meeting, Atlantic City, May 9 and 10, 1951. Pp. 296.

Modern Medication of the Ear, Nose and Throat.

By *Noah D. Fabricant, M.D., M.S., Clinical Assistant Professor of Otolaryngology, University of Illinois, College of Medicine.* Pp. xvi+245 with 20 illustrations. New York, Grune and Stratton, 1951. (Price \$5.75).

Diseases of the Ear, Nose and Throat.

By *George Portmann, M.D., Professor of Oto-rhino-laryngology at the University of Bordeaux; Dean of the School of Medicine and Pharmacy of the University of Bordeaux; Surgeon at the Hospital of Tondu, Bordeaux; Surgeon at the Hospital Leopold, Bellan, Paris.* Pp. vii+728 with 666 illustrations and 11 colored plates. Baltimore, Maryland, The Williams & Wilkins Co., 1951. (Price \$20.00)

Revista Medica da Aeronautica.

Memorial Volume Commemorating the First Decennial of the Creation of the Ministry of Aeronautics. Pp. 775, Rio De Janeiro, Brazil, 1951.

Plastic Surgery of the Nose—Including Reconstruction of War Injuries and of Deformities from Neoplastic, Traumatic, Radiation, Congenital, and Other Causes.

By *James Barrett Brown, M.D., Professor of Clinical Surgery, Washington University School of Medicine, St. Louis; Chief Consultant in Plastic Surgery, United States Veterans Administration, Washington, D. C.; Formerly Senior Consultant in Plastic Surgery, United States Army and in E.T.O., and Chief of Plastic Surgery, Valley Forge General Hospital; and Frank McDowell, M.D., Assistant Professor of Clinical Surgery, Washington University School of Medicine, St. Louis.* Pp. 427 with 379 figures, 48 in color. St. Louis, Missouri, The C. V. Mosby Company, 1951. (Price \$15.00)

Clinical Allergy.

By *Samuel J. Taub, M.D., F.A.C.P., Professor of Medicine and Chairman of the Department of Allergic Diseases, the Chicago Medical School; Professor of Medicine, Cook County Graduate School; Attending Physician, Cook County, Columbus, and Mt. Sinai Hospitals.* Pp. 276. New York, New York, Paul B. Hoeber, Inc., 1951. (Price \$4.50)

The New Way to Better Hearing.

By Victor L. Browd, M.D., *Adjunct Professor of Otolaryngology, New York Polyclinic School and Hospital; Fellow, American Academy of Ophthalmology and Otolaryngology.* Pp. xii+226 with Charts, Graphs and 8 Pages of Color Plates. New York, New York, Crown Publishers, Inc., 1951. (Price \$3.00)

1st Annual Report on Stress.

By Hans Selye, M.D., Ph.D. (Prague), D.Sc. (McGill), F.R.S. (Canada); *Professor and Director of the Institut de Medicine et de Chirurgie Experimentales, Universite de Montreal.* Pp. 511 (+133 pp. ref.) (Illustrated). Montreal, Canada, Acta, Inc., 1951. (Price \$10.00)

Reports, The Institute of Laryngology and Otology and The Royal National Throat, Nose and Ear Hospital.

Volume 1. Pp. vi+288 (Illustrated). London, Headley Bros., 1951. Price 10 shillings)

Medizinische Röntgentechnik.

By Herbert Schoen, M.D. Pp. 222 with 476 illustrations. Stuttgart, Germany, Georg Thieme, 1951. (Price DM 22.50)

First Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.

Chicago, October 1946. Pp. LII+436. Printed for the Association by William J. Dornan, Philadelphia, Pa.

Topographische Ausdeutung Der Bronchien Im Röntgenbild.

By Claus Esser, M.D. Pp. 152 with 77 illustrations. Stuttgart, Germany, Georg Thieme, 1951. (Price DM 33.00)

The Temporomandibular Joint.

Edited by Bernard G. Sarnat, M.D., D.D.S. *Chapters by Allen G. Brodie, D.D.S., Harry Sicher, M.D., John R. Thompson, D.D.S., Joseph P. Weinmann, M.D. and Arnold A. Zimmermann, Ph.D.* Pp. 165 with 62 illustrations. Springfield, Illinois. Charles C. Thomas, Publisher, 1951. (Price \$4.75)

Notices

COMPETITIVE ESSAY OF THE AMERICAN SOCIETY OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

At its annual meeting, October 30, 1951 the American Society of Plastic and Reconstructive Surgery, Inc. announced the winners of this year's awards for a Competitive Essay.

Scholarship Award: Dr. Richard B. Stark, New York City. Essay: "The Cross-Leg Flap Procedure."

Honorable Mention: Dr. Kathryn Lyle Stephenson, Santa Barbara, California. Essay: "The Production of Ectopic Cartilage."

Honorable Mention: Dr. Michael N. Tempest, Leeds, Yorkshire, England. Essay: "Cross-Finger Flaps in the Treatment of Injuries to the Finger Tip."

Honorable Mention: Dr. Allyn J. McDowell, North Hollywood, California. Essay: "Mass Treatment of Burns in Atomic Warfare."

For particulars pertaining to the 1952 Competitive Essay please write to:

The Award Committee
c/o Jacques W. Maliniac, M.D.
11 East 68th Street
New York 21, N. Y.

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct the following examinations in 1952:

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DR. CHEVALIER L. JACKSON,
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For the period of the National Emergency, the University of Illinois College of Medicine will offer a combined three year residency training program in Otolaryngology, which will include the basic course material, in fulfillment of Board requirements.

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Under this arrangement, no course fee is to be involved and in the case of most of the aforementioned institutions, a stipend is provided.

For further information, kindly address: Head of the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

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HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

(As of September 1, 1951)

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Gem Model V-60

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Micronic (Magnetic Receiver)
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Micronic Model 303
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Televox Model E
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Telex Model 99
Telex Model 200
Telex Model 300-B
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Unex Model A
Unex Midget Model 95
Unex Midget Model 110

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Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

Meeting: Toronto, Ontario, Canada, May 20-22, 1952.

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Meeting: Chicago, Ill., June 11-13, 1952.

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President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse, N. Y.

Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago, Ill.

Meeting: Toronto, Ontario, Canada, May 18 and 19, 1952.

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President: Dr. José Gros.

Secretary: Dr. Chevalier L. Jackson, 3401 Broad St., Philadelphia 40, Pa.

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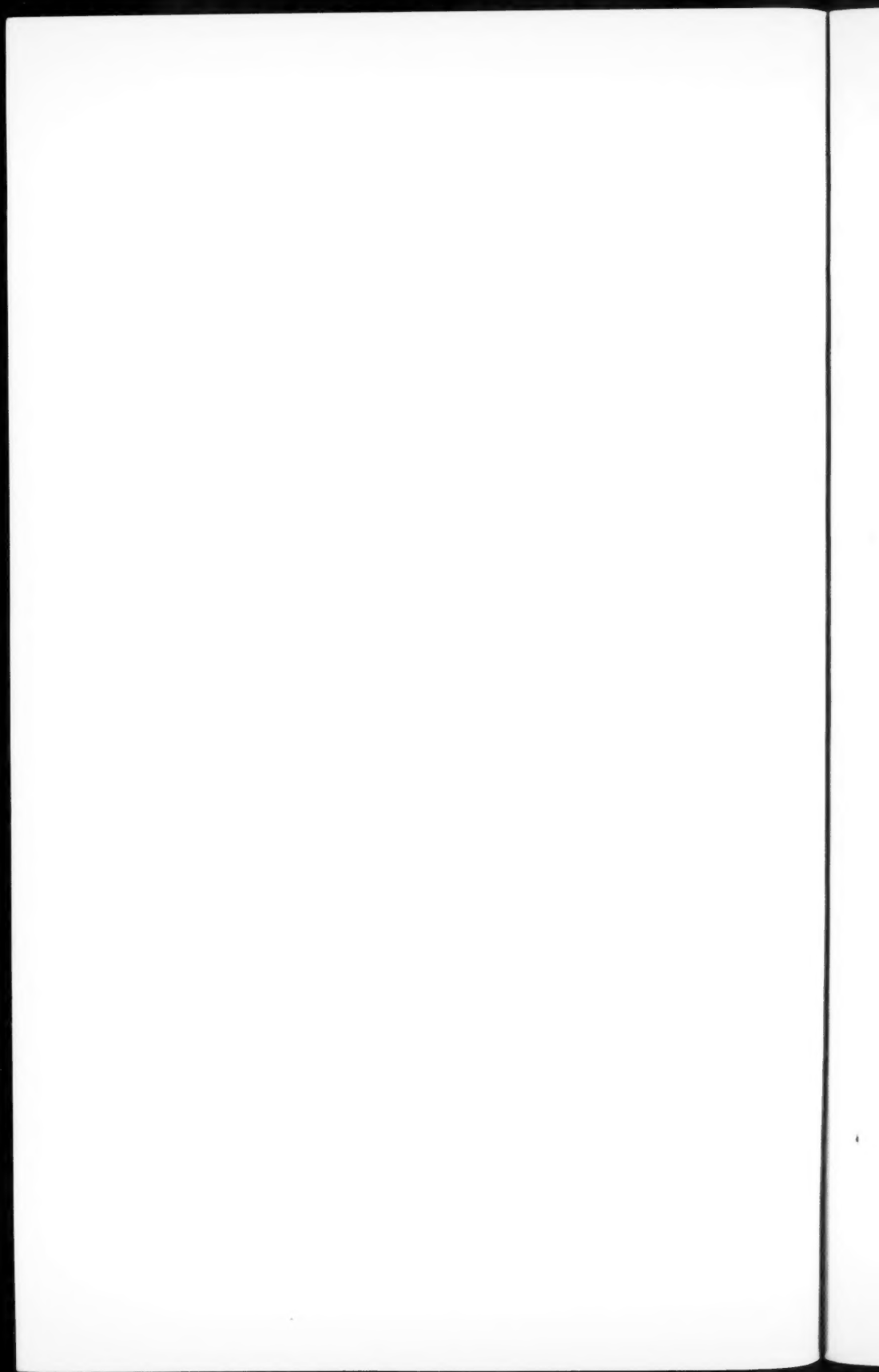
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